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GENETICS IN OTOSCLEROSIS.*

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St. Louis.

Can the inheritance of otosclerosis occur at an age level so low that a child is diagnosed as congenitally deaf? This question arose after a brief case history of a three-year-old deaf child had been taken. She was too young for reliable response to audiometer or other functional hearing tests, so if her loss of hearing has been progressive, no record could be made of it.

By personal interviews and questionnaires,† otosclerosis was traced back four generations in two families and three generations in two other families related to this child. Preceding generations had immigrated from Europe, and the living relatives had no information about their ancestors prior to their immigration.

Accurate information about auditory defects of a family is very difficult to obtain for the following reasons:

1. Information about physical defects is meager and rarely passed down from generation to generation. An individual may know if his great-grandparents had normal or defective hearing, but he rarely can describe the nature of the auditory defect, the age of onset, the extent of the impairment, or the symptoms.

2. Families seem to feel that deafness, if it is inherited, is an abnormality that should be kept a family secret and they resent answering questions concerning it.

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*Presented at the Sixty-Ninth Annual Meeting of the American Otological Society in Detroit, May 29, 1936.

†A copy of the questionnaire is appended to this paper.

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3. Humans rationalize about causes of auditory defects. If there has been illness or an accident, it will be assigned as a probable cause of deafness rather than report a similar case in family history.

4. Self-deception of patients is marked. Unless the defect in hearing is serious enough to interfere with social contacts and educational progress, they report normal hearing.

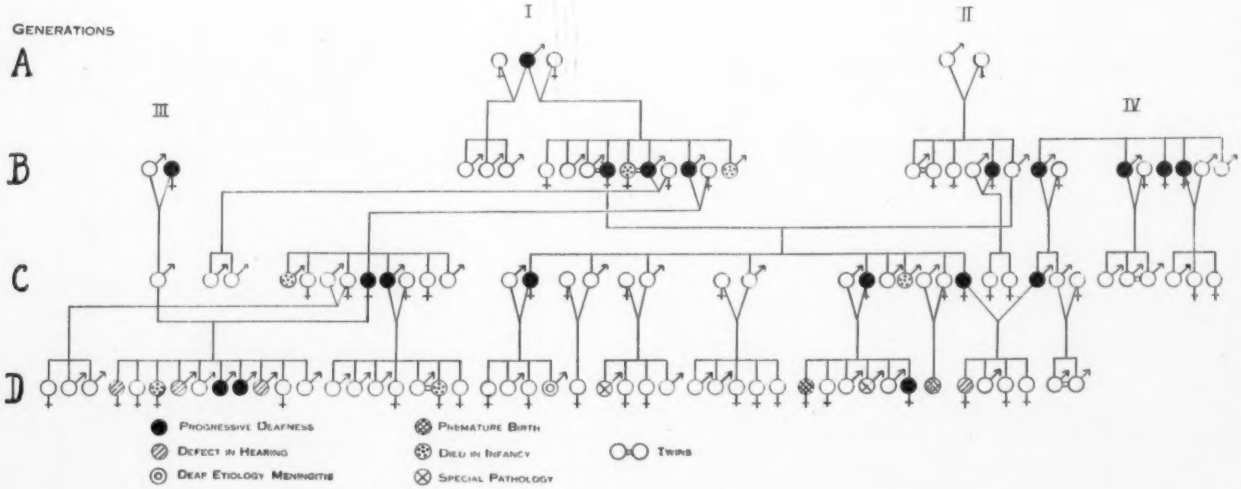
5. Accurate medical reports are rare. Otosclerotics frequently accept their gradual impairment of hearing without consulting a physician, and, therefore, there are no audiograms to show extent of loss; there are no reports of age of onset, possible etiology, or symptoms. In a genealogical study it must also be remembered that the audiometer is a comparatively new recording device, and thus audiograms of earlier generations would have been impossible to obtain.

The above genealogical table shows the incidence of impaired hearing in the four families studied and the interrelationships of these families. In the discussion of case histories that follows, reference will be made to the individuals on the Genealogical Table in the following way: The Roman numerals I, II, III, IV refer to the families; the letters A, B, C, D refer to the generations; and the Arabic numbers refer to the individuals in the family in order of birth. For example, I-III-C-3 would refer to a person in the third generation born third in the specific family which resulted from the marriage of Families I and III.

I-A-1 represents the first case of impaired hearing. He was born in Tennessee and was of German descent. His first employment was in a factory manufacturing axes, and later he worked in a blacksmith shop. He reported defective hearing, which became progressively worse as he grew older. This hearing difficulty was attributed to the constant, intense noise of his employment, particularly in the blacksmith shop. I-1 married twice. Three sons were born of his first marriage and all had normal hearing prior to the time they moved from the community as adults. Their families could not be located for a questionnaire study.

The second wife of I-1 also had normal hearing throughout her life. There were eight children born of this marriage. The oldest daughter (I-B-1) married and had six children (four boys and two girls) and no hearing difficulties are reported for either mother or daughter. One daughter died of tuberculosis after she was an adult.

A GENEALOGICAL TABLE OF AN OTOSCLEROTIC FAMILY OF FOUR GENERATIONS



Schick-Goldstein — THE LARYNGOSCOPE, Oct., 1936. FIGURE 1.

I-B-2 had perfect hearing throughout his life, and his four children had normal hearing.

I-B-3,4 were twins, a boy and a girl. The boy showed normal hearing, married, and his six children (three boys and three girls) also had normal hearing. The girl married and had nine children. She began to notice a defect in hearing when she was 21 years old, after the birth of her first child. Her hearing impairment grew worse with each pregnancy. At the age of 45 years, she was critically ill with a liver pathology, and this illness seemed to destroy the last remnants of hearing. Throughout her life she had chronic nasal catarrh, and her death was due to heart and liver complications.

I-B-5,6 were also twins. The girl died in infancy. The boy married and has two sons, both with normal hearing. When I-B-6 was interviewed, he admitted reluctantly that he can no longer hear the conversation of a group. The quality of his voice and the intensity of voice needed to make him understand, indicated a far greater impairment than he reported. During his youth, he suffered from severe nasal catarrh that has recurred at various times throughout his life.

I-B-7 was totally deaf, according to reports of relatives, at the time of his death. No one knows the age of onset or any of the symptoms of his deafness, but at the age of 40 years he could no longer hear conversation, and his deafness was progressive. He also suffered from nasal catarrh throughout his life.

I-B-7 married twice. There were seven children of the first marriage. After the death of his wife, he married again and three children were born of this marriage. His second wife left the community and took the children with her. There was no address left in this community and, therefore, no check could be made of the hearing of these children.

I-B-8, the youngest son, died in infancy.

Family II came to America from Germany. Normal hearing is reported for both mother and father in generation A. There were five children born of this marriage. II-B-1 and II-B-2 were twins with normal hearing. II-B-3 was a boy with normal hearing.

II-B-4, a daughter, had impaired hearing. No information could be obtained concerning the age of onset, symptoms, or extent of impairment. One of her otosclerotic nieces described

her as "suffering from a different kind of deafness from ours," but she could not tell how her aunt's deafness differed from otosclerosis. Throughout her life she tried many kinds

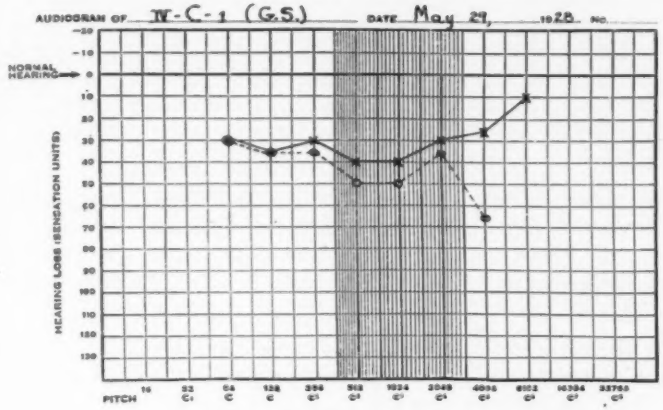


FIGURE 2.

of hearing aids, but found none of them satisfactory. She married and had two daughters, both with normal hearing. One died at the age of 18 years from tuberculosis.

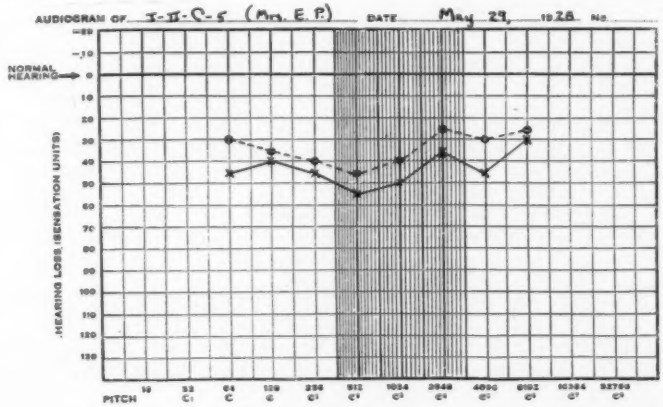


FIGURE 3.

II-B-5 had normal hearing throughout his life. He reported a severe attack of bronchitis every winter. He married I-B-4,

the twin, who reported impaired hearing after the birth of her first child.

Family III could only be traced back three generations. The father had normal hearing, but the mother had an impairment in hearing that grew progressively worse. She claimed that an otologist diagnosed her deafness as due to "a growth in one ear," but no medical record could be found, no evidence of treatment, and none of the relatives knew the name of the doctor who made the diagnosis. Their only son had normal hearing.

Family IV could also only be traced back three generations. There were five children. IV-B-1, the first son, showed all the symptoms of otosclerosis. His deafness started gradually and became progressively worse until he died. He married a woman with normal hearing and they had two sons.

The first son, IV-C-1, noted the onset of deafness just five years prior to this interview, when he was 27 years old. He reports all the symptoms of otosclerosis. He has tinnitus, which he describes as a buzzing, humming, and ringing in his ears. He also reports paracusis and his hearing impairment is becoming gradually worse. He suffers from sinus infection repeatedly. IV-C-1 married I-II-C-8, who is also otosclerotic. His audiogram is included in this paper (see Fig II).

The younger son, IV-C-2, has normal hearing. He is married and has twin boys.

IV-B-3, a daughter, also showed gradual impairment of hearing. She married, but had no children. Since she is no longer living, and never lived in the community studied, this information was obtained from the verbal reports of relatives.

IV-B-4 reports progressive deafness with tinnitus, decribed by her as an intermittent roaring. Her impairment seems to have affected low-, rather than high-pitched tones. She has three children, but does not feel that pregnancy increased her impairment of hearing. She has colds frequently and at these times head noises are more pronounced. All of her children (ages 37, 32 and 28 years) have normal hearing.

IV-B-5, the youngest son, did not respond to the questionnaire and is no longer living in this community. His nephew reported normal hearing for him, and his nephew's wife reports slight deafness. Since the reports were contradictory

and could not be verified, his hearing is represented as normal on the genealogical table.

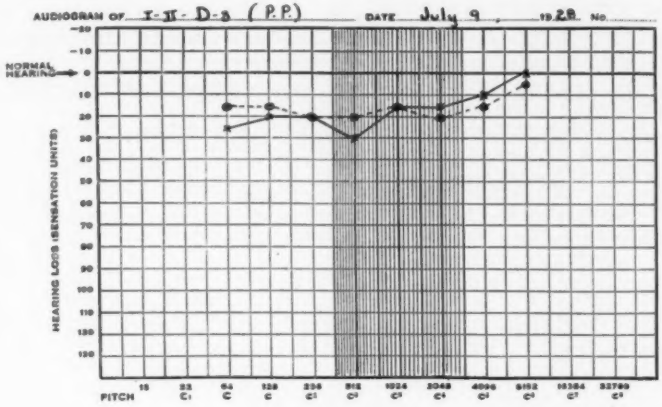


FIGURE 4.

I-B-7, who became progressively deaf, married a woman with normal hearing and they had seven children. I-C-1 died in infancy. I-C-2, a daughter with normal hearing, married,

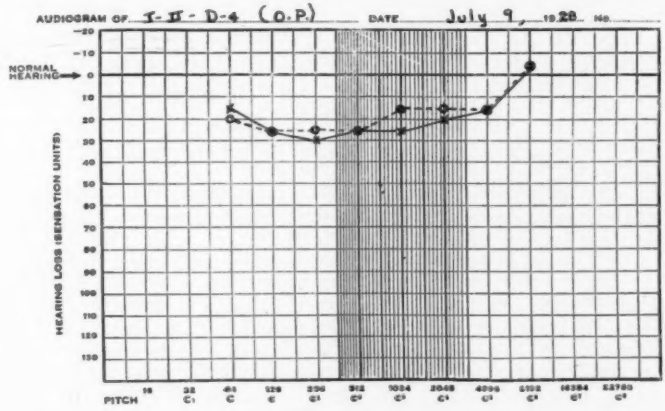


FIGURE 5.

but has no children. I-C-3, another daughter, married and had three children. She died soon after the birth of her third child. All of the children have normal hearing.

I-C-4 has progressive deafness. She married III-C-1, the only son of Family III, who had normal hearing. I-C-4 can never remember hearing normally with the left ear but claims to hear by bone conduction on this side. As a child she had scarlet fever and abscessed ears but does not feel this illness caused her deafness. She reports paracusis and an additional loss of hearing with each pregnancy—and she has 10 children.

I-C-5, a son, reported normal hearing and lived too far for a personal interview. His sister (I-C-4) said that at the time of the World War, medical examination showed his hearing to be so defective he could not be accepted for active service. Since that time she feels that his impairment in hearing is becoming gradually worse but as long as it does not interfere with his work on the farm he will not admit any deviation from normal in acuity of hearing. He is married and has seven children, all with normal hearing. The girl of a pair of twins died in infancy.

I-C-6, a daughter, died from an unknown cause as a child. Her hearing was normal to the time of her death, according to reports of her relatives.

I-C-7, a boy, was killed by a runaway horse at the age of 17 years. If there was any evidence of otosclerosis in the last two children of this family, they did not live long enough for it to develop and be noticed.

The next family to be considered resulted from the marriage of I-B-4, who was otosclerotic, and II-B-5, who had normal hearing but otosclerosis in his immediate family. They had nine children, who will be discussed in the order of their birth.

I-II-C-1, a daughter, had normal hearing until after her marriage and the birth of her first child. She has four children and her impairment of hearing has increased with each pregnancy. Repeated sinus infection seems also to increase her deafness. She reports paracusis but no tinnitus. When riding in a car or whenever the generator for their lighting system is in operation on the farm she hears better. I-II-C-1 has never had abscessed ears. Her teeth were so poor that all were extracted before she was 23 years old. All four children teethed young but have poor teeth.

All of her children, aged 28, 21, 16 and 12 years, but the youngest have normal hearing. The etiology of the deafness of the youngest boy was spinal meningitis. He has picked

up lip-reading without instruction but his speech is rapidly losing clearness.

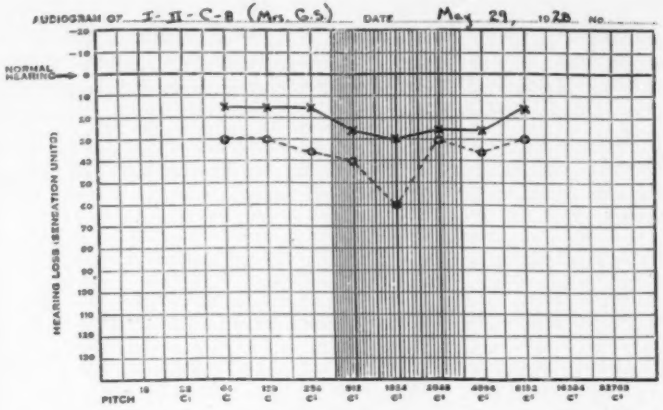


FIGURE 6.

I-II-C-2, a son, has normal hearing. He is married and has one daughter (age 20 years) whose hearing is normal also. At one time he said he showed all the symptoms of hay fever.

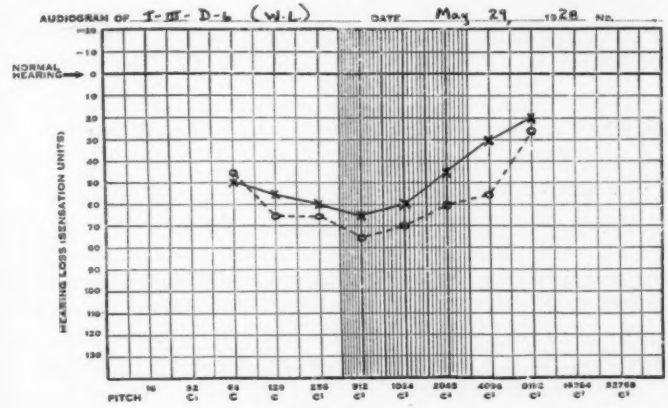


FIGURE 7.

After a physician removed "a growth from his nose," he no longer noted these symptoms.

I-II-C-3, a son, has normal hearing. Every winter he has a bronchial infection bordering on pneumonia. He married and had four children. The eldest son was a cripple from birth. The delivery was difficult and the child may have suffered a birth injury. He was described as normal in height and normal mentally but, due to a deficiency in bone structure and a lack of muscle tonicity, he was helpless until the time of his death, when he was 7 years old. The other three children have normal hearing, although the second daughter (age 11 years) has had abscessed ears several times. The youngest child, a son, was only 5 years old at the time of this interview.

I-II-C-4 has normal hearing. He married and has five children, three girls and two boys, ranging in age from 5 to 18 years. All of the children have normal hearing.

I-II-C-5 (see Fig. III for audiogram) reports all the symptoms of otosclerosis. As a child she frequently had colds accompanied by discharging ears. Her hearing was normal until after the birth of her first child. She reports tinnitus, paracusis and a progressive loss of hearing. She is very susceptible to colds which make her hearing worse, and she has had pneumonia three times. A constant ringing in her ears is described as "not steady but resembles escaping steam." Her teeth are poor, not notched, and many have been filled. The accompanying audiograms show the extent of loss of hearing.

The first child of I-II-C-5 was born prematurely and died at birth. The second child, a daughter, 18 years old, has normal hearing. Her speech, however, lacks inflection and the tempo is very slow.

I-II-D-3 (see Fig. IV for audiogram) is a boy, 17 years old. When 3 years old he fell from a barn loft. A facial paralysis was noticed on the left side when he was 5 years old and is attributed to this fall. His speech is defective, due to this paralysis. An audiogram which accompanies this study, was made in 1928. During the winter of 1931-32, he complained of continuous earaches.

I-II-D-4 (see Fig. V for audiogram), a boy, 15 years old, reported normal hearing and speech. He was operated for mastoid when he was 5 years old. His audiogram also accompanies this study.

The youngest son (age 11 years) was in excellent health and had no hearing defects.

I-II-D-6 is a deaf girl. At the time of her birth all three of her brothers had scarlet fever. The infant, born at home, had a slight rash but no fever when only a few days old. It was thought that this was a form of scarlet fever that destroyed her hearing. Throughout the entire first year of her life both ears ears were discharging.

All of these children are susceptible to colds, and none of them had their tonsils removed at the time of this study.

I-II-C-6 is a son with normal hearing who has never married.

I-II-C-7, a daughter, married and had one child born prematurely. The child died soon after birth. Her hearing is normal, but a severe jaundice has left her in a weakened physical condition.

I-II-C-8 (see Fig. VI for audiogram), the youngest daughter, is quite hard of hearing, particularly in the left ear. She reports a roaring in her ears from childhood, accompanied by a repeated discharge from the ears. In 1920, the night after a tonsillectomy, one ear discharged. About four days later, the other ear discharged and she was totally deaf for two weeks. The right ear, she believes, cleared up completely, but the hearing of the left ear has never been normal since that time. The roaring in her ears increases with fatigue or nervousness. She also has nasal catarrh, sinus infection, and poor teeth.

I-II-C-8 married IV-C-1, who also is otosclerotic. Their audiograms accompany this report. They have four children, ranging in age from 2 to 9 years. With each pregnancy the mother has noticed an increase in the impairment of her hearing.

The oldest daughter (I-II-IV-D-1) who was 9 years old at the time of this study, describes roaring sounds in her ears. As an infant she also had discharging ears. No examination of her hearing has been made.

The family of III-C, who had normal hearing but whose mother was deaf, and I-C-4, who shows progressive deafness, should be described next.

I-III-D-1, a daughter, 28 years old, was hard-of-hearing as a child but reports no noticeable defect at present. As an adult she had some difficulty with her gums and teeth that

necessitated treatment at a hospital. She is married but has no children.

I-III-D-2, a daughter, 26 years old, reports normal hearing and excellent health. Her only illness as a child was diphtheria. At present she is a graduate nurse.

I-III-D-3 died in infancy. I-III-D-4, a son, aged 20 years, noticed an impairment in hearing. A physician removed some hardened cerumen, and he says his hearing is "improved."

I-III-D-5, a boy, 18 years old, has normal hearing.

I-III-D-6, a son, 14 years old, has an impairment of hearing that becomes much worse with colds. From early infancy to the time of this study he had abscessed ears frequently. Three tonsil operations have been performed in the hope of improving his hearing, and he believes each gave only temporary relief. The accompanying audiogram (see Fig. VII) shows the extent of his loss.

I-III-D-7, a son, 11 years old, also has defective hearing. A tonsil operation is believed to have given temporary relief, but his hearing impairment is becoming progressively worse and interferes with his progress in school.

I-III-D-8, a boy of 9 years, also has defective hearing. His tonsils were removed in the hope of improving his hearing, but the operation had no effect on his auditory acuity.

The two youngest children, a girl and a boy, aged 6 and 3 years, respectively, had normal hearing at the time of the interview.

An attempt was made to determine the possible causes of otosclerosis in this community. Almost all of the cases reported live on farms. The water supply is obtained from wells or natural springs. They depend on their own produce for food, which means vegetables, fruit, and meats raised on the farm. Since the water supply is not the same for all of the families studied, and since the food supply varies with the products raised, it does not seem likely that these external conditions have seriously influenced the development of otosclerosis.

There are no instances of consanguinity in any of the families or generations studied.

The physical condition of this group may, however, have some relation to the frequency of occurrence of progressive deafness. Regardless of whether hearing is normal or defective, almost all members of these families report high sus-

ceptibility to colds, sinus infection, and nasal catarrh. The percentage of poor teeth seems higher than would be found in the average family group. There is one case of pathology of bone structure not associated with this form of deafness.

Almost all of the families are above average in size. In generations B, C, and D of Families I, II, and IV, there are six pairs of twins. This genealogical table also shows two premature births and eight cases of death in early infancy. Perhaps both the frequency of twinning and the frequency of infant mortality are hereditary or chance traits that have happened to occur in the same families and are unrelated to the auditory impairment—but both of these observations seem to merit further investigation in other otosclerotic families to affirm or deny a relationship.

This family study was made in the interest of the girl (I-II-D-6), now a pupil of Central Institute for the Deaf, who seems to show total deafness. At the time of the study an accurate hearing test with tuning forks, audiometer, etc., could not be made because she was too young (3 years). Is her auditory defect of the progressive type that appeared at a very early age due to the cumulative effect of three generations of otosclerosis in her family? This question will have to remain unanswered.

GENETIC CONCLUSIONS.

1. Deafness occurs in all four generations, an unusual condition in hereditary transmission. No previous genetic studies of otosclerosis have been traced back more than three generations.

2. When defective hearing occurs on both sides of the family there is: *a.* An increase in the per cent of cases. *b.* Defective hearing at a lower age level.

3. There seems to be a relationship between other physical ailments, such as susceptibility to colds, sinus infection, nasal catarrh, and poor teeth and progressive deafness. What this relationship may be has not been determined.

OTOLOGIC CONCLUSIONS.

The detailed investigation of this extensive otosclerotic family, with many ramifications, serves a manifold purpose:

1. Evidence that otosclerosis is an hereditary disease of the ear and may be transmissible, generation after generation, without a break.

2. That otosclerosis may develop at a very early age and that its insidious onset may be overlooked and may not be easily diagnosed.

3. That otosclerosis, of marked degree, occuring at very early age in unusual onset, may simulate congenital deafness.

4. There are no Wassermann tests accompanying this study, but there is no evidence in the familial reports, clinical examination of the patients concerned in this investigation, and no data of functional tests or audiograms pointing toward marked nerve deafness. Therefore, the possibilities of syphilis as a genetic factor are minimized.

5. That there is *no* justification, by evidence produced in a great number of cases, that congenital deafness is an hereditary disease.

6. That there *is* justification for the consideration of otosclerosis as an hereditary disease.

7. That there is a possibility that otosclerosis may be developed in embryo and that such embryonic otosclerotic pathology may be confused with and show aspects of similarity to congenital deafness.

These observations are the result of this extensive survey of a detailed familial history of four generations and may offer data for the further development of a field which may throw some light on the origin of both congenital deafness and otosclerosis.

DISCUSSION.

DR. J. GORDON WILSON:

This excellent genetical report, painstaking and thorough, is a study of the incidence of otosclerosis in four families carried back four generations. Much of the data was obtained from personal examination of the patients and from interviews with relatives; in addition, when personal contact was not possible, by a questionnaire. Information so obtained is acknowledged to be of greater value than the information elicited during a routine office examination.

The importance of genetic studies cannot be gainsaid. Heredity is an undisputed factor in the causation of otosclerosis. We may grant that it cannot always be traced, a circumstance not to be wondered at for many reasons, including the obvious one that it is on a particular localization of the lesion that diagnosis depends. It is acknowledged that factors other than heredity play a part. Here, consanguinity, always important in heredity transmissions, is lacking. Since these families lived on farms there was good opportunity to study the effect of soil and water. No such influences could be traced. It is striking how frequently nasal and postnasal catarrhs come into the picture, a fact noted also by Gray.

DR. STACY R. GUILD:

Dr. Goldstein has submitted a very fine genetic study. I have been very much dissatisfied, as all of us have been, with the usual type of report in this field.

In our collection of sectioned temporal bones, the majority of otosclerotic foci found do not involve the footplate of the stapes. In other words, otosclerosis may be present histologically, but we cannot recognize the condition clinically, and the patient is not deaf unless the footplate is involved. This is not a new observation. I said it six years ago and it is widely recognized as a fact.

Therefore, in a genetic study, is it not entirely possible, indeed probable, that many of the members of a family who are not clinically deaf do have in their temporal bones a focus of otosclerosis which has not chanced to invade the footplate of the stapes? It is an accident whether or not the annular ligament or footplate is involved; the area in front of the oval window is the real place of predilection for otosclerotic foci.

The other thing is: In all genetic studies one speaks of sporadic cases of otosclerosis or cases without a positive family history of deafness. We do not know whether or not there is in that family line an histological otosclerosis which crops out into clinical recognition at rare intervals by an "accidental" involvement of the footplate of the stapes.

QUESTIONNAIRE USED IN THIS INVESTIGATION.

Name .....

Date of birth .....Sex.....

Date of marriage.....No. of children.....

Give ages of your children and their sex.....

.....

Have you an impairment in hearing? YES NO

When did you first notice the impairment?.....

Have you tinnitus (do you hear continuous or intermittent noises in your ears)? YES NO

Underscore the words that best describe these noises: Buzzing, hissing, humming, ringing, roaring, rumbling, tinkling.

Have you paracusis (ability to hear better in the presence of sounds like the hum of a motor, or when riding on train, street-car or auto)? YES NO

Can you hear a soft, high-pitched whistle (like escaping steam) as well as conversational voice? YES NO

Can you hear low, humming sounds as well as conversational voice? YES NO

Is the impairment in your hearing gradually becoming worse? YES NO

Has your impairment in hearing become worse after pregnancy? YES NO

Were any children born prematurely? YES NO

Underscore kind of birth, using one line for each child: Natural, prolonged, forceps delivery, Caesarian.

Did any children die in early infancy? YES NO

If so, what was the cause of death?.....

Underscore any of the following diseases you have had: Chickenpox, diphtheria, measles, mumps, scarlet fever, typhoid, whooping cough.

List any diseases you have had that are not mentioned in the list given above .....

How soon thereafter did you notice hearing impairment?.....

.....

What is the state of your general health? Good Fair Poor

Underscore any of the following conditions you have had: Frequent colds, chronic nasal catarrh, sinus infection, hay fever, recurring sore throat, tonsillitis, earache, running ears, adenoids, mouth breathing, bad teeth, anemia, rickets, bone injuries.

Has a Wassermann test ever been made? YES NO

What were the results? Positive Negative

Is there any tuberculosis in your family? YES NO

Are your parents related? YES NO

What is the relationship?.....

Have there been twins in your family? YES NO

How are they related to you?.....

Are there any physical disabilities, other than impaired hearing in your family? YES NO

Underscore any of the following disabilities present: Blindness, defective vision, paralysis, lameness, spasticity.

Has an ear specialist been consulted about your impaired hearing? YES NO

Give name and address so we may write him for further particulars.

.....

Are there other cases of impaired hearing in your family? YES NO

Underscore the relatives who have impaired hearing: Paternal grandfather, paternal grandmother, maternal grandfather, maternal grandmother, father, mother, brother, sister, paternal uncle, paternal aunt, maternal uncle, maternal aunt, husband or wife, son, daughter, niece, nephew, cousin.

NOTE: The purpose of obtaining this information in questionnaire form is to afford an opportunity for an accurate survey and study on a large scale of all types of hearing disabilities, so that we may be in a position to organize ways and means of overcoming these handicaps.

If any of your relatives have impaired hearing, will you please give their names and addresses so that we may request them to fill out such a questionnaire? If it is not possible to obtain this information from them directly, would you fill out a similar questionnaire as accurately as you can for them? YES NO

	Name of relative	Address
1.	.....	.....
2.	.....	.....
3.	.....	.....
4.	.....	.....

818 South Kingshighway.

THE SELECTION AND OPERATION OF AUDIOMETERS.*

E. G. WITTING, Ph.D., Abington, Pa.

At the present time the question of tuning forks versus audiometers for auditory measurements is still in the controversial phase. Although audiometers have not been unanimously accepted, there has been a marked increase in their popularity, resulting in the appearance of several new types. The design of audiometers is a complicated process, executed by persons in a specialized field and featuring many points with which the average otologist is not expected to be familiar. Realizing this, it would seem that information intended to acquaint prospective purchasers with some of the salient "purchase points" might be of value.

It is difficult to set aside certain definite constituents of the audiometer in iron-bound order of importance since so much coordination exists in the functioning of the instrument; however, in the following, an attempt has been made to group the points in a somewhat loosely bound order. The following are considered: Tone purity; adequate functioning of attenuator; air conduction receivers; extraneous noises during operation; frequency; bone conduction receivers. In addition to these points, certain suggestions are made concerning operating technique.

There are three essential units of an audiometer, a tone generator, a control for reducing the intensity of the tone, and a telephone receiver for reproducing the sound. The purity of the tone (freedom from overtones) is very important, carrying more weight at the low than at the high frequencies. When a tone is generated, either electrically or mechanically, overtones which may be quite appreciable almost inevitably accompany the desired frequency. A good audiometer must provide reasonably pure tones. A brief discussion

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of the hearing sensitivity of the human ear may serve to strengthen this requirement.

Measurements[1] have shown that the sensitivity of the normal ear is approximately 15 decibels greater at 128 cycles than at 64 cycles. Hence, it is quite obvious that in a 64 cycle tone, which is so impure that the 128 cycle overtone is just 15 decibels less intense than the fundamental, the average normal ear will hear the two components with equal ease; however, if the 128 cycle overtone is less than 15 decibels lower in intensity than the 64 cycle fundamental, the ear will hear the 128 cycle component better, and when the frequency is attenuated to threshold for measurement of hearing the 64 cycle component will become inaudible, while the 128 cycle component is still being heard. Hence, the measurement of hearing will not be the response to the nominal frequency, 64 cycles, but the response to the harmonic contained in the impure tone. This calculation is based on the normal ear. If a subject's hearing is actually 5 or 10 units better at one of the overtones of the nominal testing frequency than at this frequency, then the overtone in the frequency definitely becomes the dominant factor. For frequencies above 2000 cycles per second, the sensitivity of the ear decreases and hence, overtones cease to be quite as disturbing. If an analysis of the wave shape of the tone generated by the audiometer and emitted by the receiver has been made, the following figures may serve as an approximate maximum harmonic requirement:

64 cyles—128 cycle harmonic intensity at least 30 decibels lower than fundamental; other harmonics at least 50 decibels lower than fundamental.

128 cycles—256 cycle harmonic intensity at least 25 decibels lower than fundamental; other harmonics at least 35 decibels lower than fundamental.

256 cycles—512 cycle harmonic intensity at least 20 decibels lower than fundamental; other harmonics at least 30 decibels lower than fundamental.

512 cycles—1024 cycle harmonic intensity at least 15 decibels lower than fundamental; other harmonics at least 25 decibels lower than fundamental.

1024 cycles—2048 cycle harmonic intensity at least 15 decibels lower than fundamental; other harmonics at least 25 decibels lower than fundamental.

If no analysis is published the presence of overtones near threshold intensity should be tested by listening.

Instruments operating on alternating current supply can contain hum which may vitiate measurements at the higher frequencies. If extreme care is not exercised in removing the hum from the rectifier which supplies voltage for the operation of the tone generating vacuum tube, it will be heard by patients with high tone losses but good low tone response. In such cases it is almost impossible to elicit a response from the patient for the high frequency only; the result being that the patient's hearing is measured for the low frequency hum and not for the frequency of test. Detection of this is somewhat difficult, but the audiometer is reasonably safe if the hum threshold is at least 50 decibels below the normal threshold for all frequencies above 8192 taken on the ear of a person normal at the low tones. This simple test can be made easily by any normal hearing person. In audiometers providing sweep-frequency change, subfrequencies, *i. e.*, tones below the nominal frequency, may be generated. These also become evident at the high frequencies and their presence can be tested by setting the audiometer for a high intensity and sweeping the frequency backward and forward through the high frequency range. The subtones are most likely to appear for frequencies above 10,000 or 12,000 cycles and their effect is similar to excessive hum.

The tone generated by an audiometer, unlike a tuning fork, is an undamped sound, hence, means must be furnished to reduce its intensity to threshold of audibility. This is accomplished by providing a network of resistances which attenuates the electrical voltage before it is applied to the receiver. The voltage applied to the receiver for the loudest sound is approximately one million times that for the lowest sound and in order to provide adequate intensity range for measurement, the attenuator must include sufficient attenuation to cover this range. In attenuators incapable of handling this large range properly, the sound reaches a minimum for a certain attenuation and cannot be reduced further when the attenuator dial is turned to full attenuation. Obviously, this is of no value in measuring patients normal or slightly better than normal in one ear. This type of defect can be detected by a person of normal hearing listening to the audiometer in a soundproof room or in a very quiet room.

The hearing loss scale deserves some consideration. Unique scales should be avoided. It is probable that the sensation unit

or decibel scale will be standardized, because of its general acceptance at the present time.

From the attenuator the current is passed to the final element, the receiver, which probably equals the tone purity in importance. It would be highly desirable for manufacturers to use receivers of a type which would be entirely uniform in response from receiver to receiver (to a small allowable variation). In this way, if the audiometers were built to conform to a standard output, two different audiometers in separate offices would give identical results on the same patient; however, this is a difficult requirement to meet and probably the most to be expected is that the response of the receiver on a given audiometer remain constant to an amount less than the intensity step of the intensity control.

The question of frequency range desired can be settled by the individual otologist, and he can be guided accordingly in choosing the audiometer. The eight-frequency audiometer seems to meet general requirements; on the other hand, the additional band available in sweep-frequency instruments is valuable in certain cases. The frequencies of audiometers having discrete frequency settings should be close enough to octave steps so that the average person will not detect an unpleasant flatness as the tone is changed.

If a bone conduction receiver is used with the audiometer, the precaution should be taken that the air radiated sound is of lower intensity than the bone conduction perception as measured on an individual of good air conduction. At the present moment receivers for use in diagnosis of bone conduction have not reached a stage of development proportionate to that of the air conduction receiver. As receivers are developed and appear on the market, it would be well if they were investigated by impartial laboratories and the findings reported.

The operation of an audiometer is probably as important as the necessity of care in its choice and, although operating instructions are always supplied with the instrument, it is felt that a few suggestions might be worth consideration.

It seems reasonable to take the frequencies in order of their number, starting at the lowest and progressing upward; however, patients seem to experience more difficulty in observ-

ing the high and low frequencies than those in the middle range. Hence it is suggested that the operator start with a tone of 1024 or 2048 cycles and carry the measurement to the high frequencies, upon completion of which the measurement should be extended from the middle range to the low frequencies. At the frequencies below 256 cycles, care should be taken to insure the patient holding the receiver close to the ear. Slight leaks caused by holding the receiver partly off the ear will result in a lowering of the recorded hearing at these frequencies. It is the opinion of some otologists that an element of hearing by bone conduction is produced by holding the unprotected receiver directly on the ear. This need be of little concern, but those who feel safer with this possibility eliminated may use a soft rubber ear cap on the receiver. In selection of a cap, care should be taken that there is no undue pressure change resulting from the use of an entirely nonporous cap. Sponge rubber with a semismooth finish seems to give best results. If the audiometer is supplied with ear cap it will prove worth while to examine the cap with the above points in mind. If the audiometer has been calibrated without the rubber cap, the use of the cap will introduce an error, which may be compensated in recording if the exact effect of the cap is determined. This can be done fairly satisfactorily by measuring the hearing of two or three individuals with and without the cap under very quiet conditions, a soundproof room being preferred. If the audiometer must be used in a partly noisy location the use of a headband with both the audiometer and a dummy receiver equipped with rubber caps is to be preferred to a receiver without cap. In this case, with a reasonably good headband, a good seal is insured for the lower frequencies.

As shown by Bunch[2] wide variations are to be expected at high frequencies, due to age variations in hearing; however, there is also a physical condition which contributes toward a spread at these frequencies. Above approximately 4,000 cycles, the wave length of the sound wave is short enough to create the phenomenon familiar to most otologists and known as "standing waves." If the wave from the receiver diaphragm reaches the head at the proper portion of the wave it can be reflected so as to almost entirely neutralize the original wave. This cannot occur at the low frequencies because the wave length is so long that the proper position on the wave cannot

be attained in the short travel from the receiver to the ear. A difference of as much as 20 decibels can be observed due to this effect. This can be eliminated by moving the receiver slowly toward and away from the ear of the patient after having first obtained a reading with the receiver close. As the change in receiver distance causes the sound to go through a maximum of intensity the patient can signal. For measurements above 8000 cycles this procedure is quite essential. If removing the receiver from close contact with the ear causes the operator to suspect that the sound is heard in the opposite ear (in cases of unilateral deafness) the good ear should be masked.

Masking one ear as a means of insuring the sound being heard by the opposite ear has been in use for some time. The Barany *lärmaparat* is a familiar form of noise production apparatus. It has the disadvantage of producing a rather loud sound in the air, which can be heard in the ear under test. Also the volume of sound produced by the instrument is not subject to adjustment by the user. The committee on methods of testing by bone conduction of the American Otological Society has made recommendations concerning the best types of masking apparatus. The committee reports the following concerning the type of sound suitable for masking: "Mixed sounds, or noises, are better for the purpose for which an otologist uses masking than are pure tones. Theoretically, the ideal sound to use for masking would be an even mixture of all frequencies that can be heard, from the lowest to the highest." The use of masking is of great importance in the interpretation of audiograms and in determining the existence or nonexistence of perception in cases of unilateral deafness. This same committee reports: "When masking is not used the examiner has no choice but to accept the statement of the patient as to which ear hears the sound. The purpose of masking is to enable the examiner to use objective methods in the determination of the hearing acuity of the poorer ear as well as of the better ear." The subject of masking has been covered quite fully by Bunch.[3] In the measurement of bone conduction it is imperative that masking of some type be used on the opposite ear.

The conditions under which the hearing test is conducted go far toward contributing to its accuracy. In spite of the

fact that the patients exist in noisy surroundings, there is no advantage in measuring their hearing under such conditions. The otologist should regard the soundproof chamber for hearing measurement as a valuable adjunct to his office equipment. It can be dispensed with under certain conditions (very quiet location and use of rubber receiver cushions) but is a great aid to proper interpretation of audiograms.

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Abington Memorial Hospital.

REPORT OF A CASE OF MAXILLARY SINUSITIS WITH COMPLICATING, SPREADING OSTEOMYELITIS, BRAIN ABSCESS AND DEATH.

DR. A. NEIL LEMON, Philadelphia.

Mrs. H. P., age 27 years, came to me in October, 1931, complaining of frequent colds. There was nothing outstanding in the history. She had long hours as a hairdresser, worked hard and was 15 or 20 pounds underweight. On examination I found diseased tonsils which I later removed under local anesthesia. The sinuses were practically negative, and there was no nasal obstruction. After tonsillectomy the patient was given a course of vaccines with the hope of increasing the resistance to catarrhal infections; but the colds persisted, and she gained no weight.

In the early part of 1933 she presented herself with a severe cold, fever, and pain surrounding the left eye. The left maxillary sinus was punctured and lavaged and a large amount of pus recovered. The procedure was repeated four times during the next 10 days, the fifth washing being clear, and all symptoms had subsided. Following this there were three similar attacks during a period of six months. On frequent occasions since that time the left antrum was lavaged, and a moderate amount of yellowish-white pus evacuated.

The condition continued until May, 1935, when an X-ray examination showed the following: "Frontal sinuses entirely absent. All the posterior ethmoid cells on both sides are cloudy and indistinct. Both maxillary antra show uniform density throughout, the left being more dense. No fluid level can be determined. Sphenoids normal."

A bilateral Caldwell-Luc operation was done in Temple University Hospital under avertin anesthesia on May 24. A large amount of polyp tissue with purulent exudate was removed from both antra and each cavity packed lightly with

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iodoform gauze. After 48 hours about one-third of the packing was removed by way of the nostrils and the following day the remainder was removed. The patient was discharged from the hospital four days after operation with no fever, no visible swelling externally and apparently in good condition. On the seventh postoperative day the patient was perfectly comfortable, at which time I lavaged the operated sinuses and found nothing more than a few small blood clots. On the ninth postoperative day there appeared swelling and pain was felt over the left maxilla. Lavage of the nose and antra was clear and the temperature normal. The sites of the original canine incisions were inspected and the left one probed, evacuating a few drops of pus. A nasal shrinking solution and mild sedatives were prescribed. During the several days following, the swelling and pain over the left maxilla became more pronounced, and the patient was readmitted to the hospital June 8. On June 12, the gums became painful and a smear from same was reported positive Vincent's angina. Sodium perborate was used locally and one dose of neoarsphenamine given intravenously. Hot compresses were used over the left maxilla, the surface of which had become quite red and swollen but with no evidence of pointing. At this time the temperature was practically normal and there was little or no nasal or postnasal drainage.

On June 16, there was a spontaneous rupture through the skin about one-half inch below the left inner canthus, and from this point drainage was profuse and continuous. A smear from this showed no Vincent's organisms and culture showed staph. aureus and nonhemolytic streptococcus.

On June 23, the patient returned home after her husband signed a release from the hospital. The area over the left maxilla took on a brownish-red color and the swelling persisted in spite of profuse drainage. On June 25, Dr. William A. Steel saw the case in consultation and advised the continued use of hot compresses and watchful waiting. On June 28, there was noticed considerable edema and ecchymosis above and below the left eye. Cavernous sinus thrombosis was suspected and the patient was again sent to the hospital. Eye ground study by Dr. Gibson and Dr. Lillie showed no evidence of thrombosis. On admission the temperature was 102° and the patient was in considerable pain, opiates being necessary.

During the next few days the following laboratory reports were made: Blood count showed hemoglobin, 8:75 gm. per 100 cc.; R.B.C., 3,700,000; W.B.C., 13,000, with 74 per cent neutrophiles. Urinalysis was normal. Blood culture was sterile. Blood calcium, 9.7; and phosphorous, 3.84. Smear from face drainage showed no Vincent's organisms and culture showed B. coli. Smear from mouth showed no Vincent's organisms and culture showed nonhemolytic staphylococcus albus and

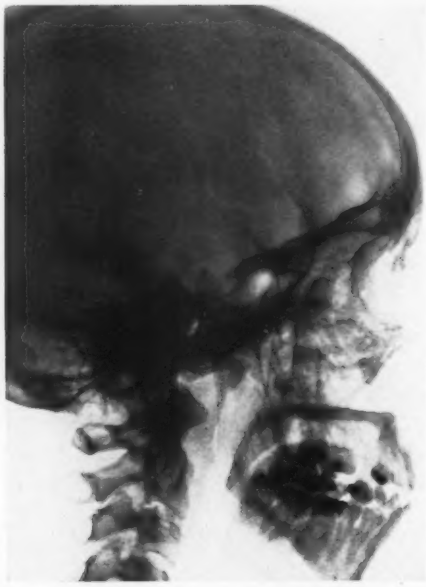

Fig. 1. Film taken July 15, 1935. Shows extensive osteomyelitis of facial bones on left side. At least two sequestra of maxilla can be seen.

diphtheroid. Spinal fluid showed 4 leukocytes per cu. mm.; was clear and under 14 mm. pressure in recumbent position. Blood Wassermann and Kahn, negative. Spinal Wassermann, negative. Before the patient was married, about three years ago, the intended husband confided to me the fact that a few years earlier he had had specific intravenous therapy and was pronounced cured. I assured him that another test should be made but as far as I know it was not done.

On July 1, Dr. Steele made a one inch incision over the left maxilla and found pus in the soft tissue and roughening of the bony maxillary surface.

Medical treatment up to this time had consisted of necessary sedation, hot boric compresses locally, glucose and Pregl's iodine intravenously, nasal shrinkage, and frequent blood transfusions. Both antra were lavaged without difficulty several times but were found clear.

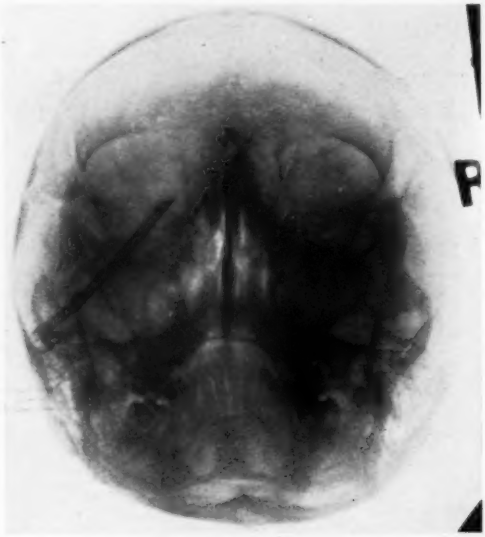

Fig. 2. July 24, 1935. Extension of osteomyelitic process to involve frontal bone also.

On July 13, Dr. Gibson incised the left lacrymal sac, which drained freely.

X-ray study on July 15 revealed an extensive osteomyelitic process involving the left maxilla, nasal and frontal bones. The left maxilla and left nasal bone showed sequestration. The temperature ranged between normal and 100°, rarely going higher.

Pain persisted through the face and forehead regions. An X-ray examination on July 24 showed slight extension of the

osteomyelitic process, mostly upward to the frontal and orbital regions. On the same day under avertin anesthesia Dr. Steel removed practically all the facial surface of the left maxilla, which had sequestrated. The malar bone, orbital plate and alveolar process were found healthy at this time. The right side was explored and the maxilla showed a lack of periosteal covering but no sequestral separation.

The patient's condition gradually became worse and on Aug. 8 an X-ray examination revealed more destruction. The

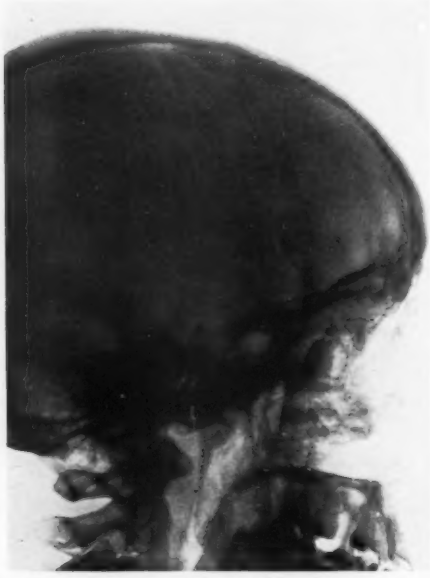

Fig. 3. Aug. 8, 1935. Continued bone destruction, including involvement of sphenoid ridge on both sides.

osteomyelitis had spread posteriorly about the orbit to involve the sphenoid ridge. On this date more sequestra were removed by Dr. Steel. On the right side the sequestra were taken from along the entire lower border of the orbit. On the left side the maxillary process, nasal process and orbital plate to one-half inch depth into the orbit were removed. Culture of the pus at this time showed nonhemolytic streptococcus, nonhemolytic staphylococcus aureus and bacillus pyocyaneus.

Under the direction of Dr. Kolmer a bacteriophage was prepared from the staphylococcine growth and administered locally in the wound and intramuscularly. Frequent blood transfusions were continued and the use of small doses of insulin was begun. This helped in promoting an appetite. Venaclysis of saline was used between blood transfusions.

X-ray study on Aug. 27 showed extension of the destructive process upward in the frontal bone and a slight increase in the backward extension of the osteomyelitis along the

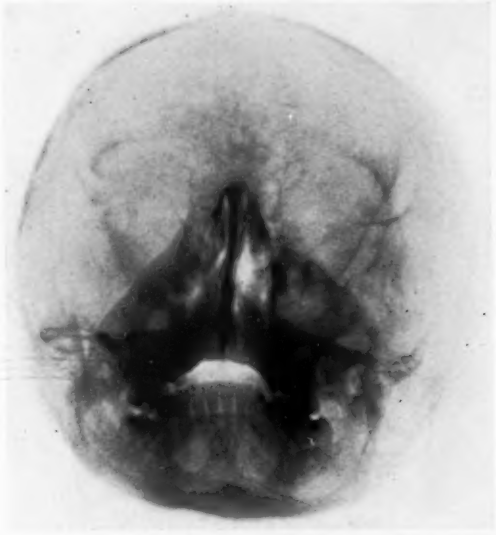

Fig. 4. Aug. 27, 1935. Continued extension. Shows absence of part of lateral aspect of left antrum, which has been removed surgically.

sphenoid ridge. During the next two weeks several superficial abscesses were opened and drained over the frontal and temporal regions. X-ray on Sept. 11 showed continued extension in the frontal bones with what appeared to be beginning sequestration just above the region of the frontal sinus. There appeared at this time some evidence of bone regeneration in the maxilla. Further study on Sept. 25 revealed a more moth-eaten appearance of the frontal bone than before with more evidence of areas of sequestration. Regeneration of bone

below the level of the orbits and along the sphenoid ridge was evident.

Realizing that conservative treatment along with removal of small sequestra as they separated was not controlling the progress of the disease, a radical procedure was decided upon. This was done on Sept. 27 and a description of the procedure is as follows: An incision was made across the vertex of the skull, exposing freely the entire frontal area to one-half

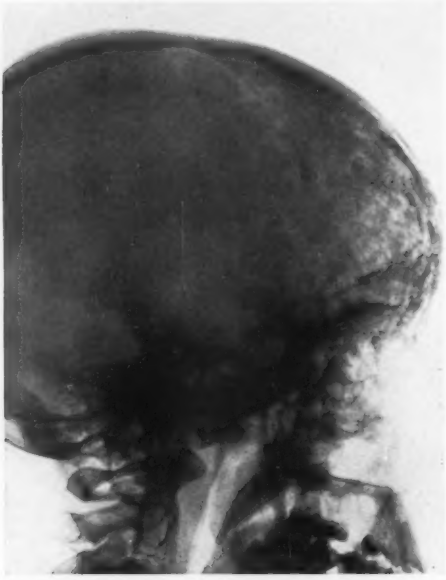

Fig. 5. Sept. 11, 1935. Extension of disease to suture between frontal and parietal bone on left. Sequestration of frontal bone.

inch posterior to the coronal suture. Multiple epidural abscesses over both temporal regions were evacuated. At the point of the anterior fontanelle the frontal bone was separated from the dura by an oblique chisel cut one-half inch anterior to the coronal suture. The whole temporal bone was removed down to the right supraorbital ridge and including the left supraorbital ridge and part of the left orbital vault. The chisel cut through healthy bone along the upper part of the frontal. The bone was then broken back and separated

at the coronal suture. The wound was closed with free drainage openings in the temporal regions and at the supraorbital ridges. The dura was punctured at points of tension over the temporal areas and a small amount of clear fluid evacuated. No subdural pus was found. A small area of brain, $\frac{1}{8}$x$\frac{1}{4}$ inch, in the left frontal eminence area was devoid of meningeal covering.

The patient's condition was poor and supportive medication was needed postoperatively. For several days following oper-

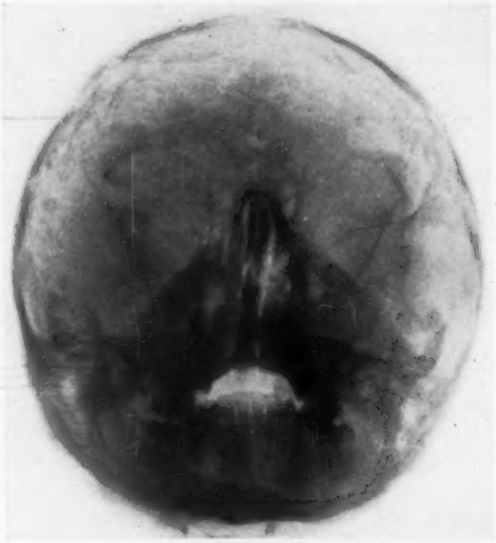

Fig. 6. Sept. 25, 1935. Moth-eaten appearance of entire frontal bone.

ation the temperature remained just a little above normal and the patient rallied somewhat. Forty-eight hours after operation she exhibited a marked degree of irritability and for periods of time was irrational, but showed no definite signs of meningitis. The left eye became more proptosed and there was occasional vomiting. On Oct. 4 it was noted that she had great difficulty in swallowing. On Oct. 5 paralysis of the left side of the face and left arm was evident. There was involuntary passage of urine and feces. Large quantities of

glucose and saline were given intravenously and subcutaneously. On Oct. 6 a superficial abscess over the right frontoparietal area was incised and drained and the following day about 1 oz. of pus was evacuated by spreading the end of the vertex incision over the right temporal region. The power of speech became very weak and it appeared that the patient knew what she wanted to say but was unable to make herself understood.

On the following day, Oct. 8, under local anesthesia the following steps were taken: The original suture line across the vertex was laid open along the whole length except at the midpoint. Lateral cuts were made in each temporal region. The dura was incised over the fissure of Rolando on the right side and about 4 oz. of pus evacuated. The abscess extended down over the right frontal lobe. An epidural collection of 1 oz. of pus was evacuated in the left temporal region. A small piece of parietal bone was broken off, revealing healthy bone with no evidences of osteomyelitis. Free epi and subdural drainage was inserted. The patient came out of the stupor after the subdural abscess was evacuated and talked rationally on the operating table. A trocar puncture made through the dura in the left temporal region to a depth of three-fourths inch into brain substance revealed no pus.

The patient was returned to her room without closing the wounds but with adequate rubber drainage. Her condition became worse although she was more rational. Two days after the subdural abscess was evacuated there was noted a cerebral herniation through the incised dura. This herniation became larger each day and every exposed area was bathed in pus. No further heroic measures were instituted and the patient succumbed on Oct. 17.

In reviewing the reports of 91 cases of osteomyelitis of the frontal bone as a complication of sinus disease we find the following statistics:

The average age is 25 years. It is noted that of these, 67 per cent were between the ages of 15 and 30 years.

As to sex, 60 per cent were male and 40 per cent female.

The predominating organism was staphylococcus aureus.

Forty-two per cent of the cases complicated acute frontal sinusitis, 35 per cent complicated chronic frontal, 1.5 per cent

followed acute maxillary sinusitis and 6 per cent from chronic maxillary; 8 per cent came from pansinusitis.

The mortality was 51.6 per cent and of these, 55 per cent followed an acute infection and 45 per cent followed a chronic infection.

In the cases resulting postoperative the average interval between the sinus operation and the onset of the osteomyelitic process was 10 days.

The average number of surgical procedures for eradication of the osteomyelitis was four.

The average interval between onset of osteomyelitis and either death or recovery was 13 months.

Brain abscess was a complication in 30 per cent of the cases.

General treatment consisted of removal of sequestra as they separated; drainage of all superficial infections as they arose; blood transfusions; bacteriophage and vaccines; colloidal silver intravenously; and appropriate supportive measures.

The following statements can be made as representing the thoughts of the majority of the authors on the subject:

1. The infection travels in the diploe between the tables of the skull and is always further advanced than can be demonstrated by X-ray.

2. Mortality is high at best but is higher in postoperative cases.

3. Osteomyelitis complicating sinus surgery is not as self limited as that which occurs spontaneously.

4. The best results are obtained by radical resection of the bone well beyond the limits of disease.

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LINGUAL THYROID.*†

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The purpose of the thesis is to present an analysis of the cases of lingual thyroid or goitre reported to date, together with a citation of representative articles dealing with the embryology, the anatopathologic features, and the treatment of the condition. Finally is added a personal case with clinical history and photomicrographs of the sections from the tumor.

INCIDENCE.

Lingual thyroid or goitre has been the subject of complete treatises, notably at the hands of continental writers. Doré, in 1922, is generally credited with having brought the literature completely to that date with a total of 80 cases, his own making 81. Apparently, no one subsequently had the trouble to search the literature and this figure has been generally accepted. It is possible, however, to identify between Hunt's report in 1865 and Doré's thesis in 1922, 91 additional case reports or references to cases in discussions of recorded cases, a total of 172, while in the same year, 1922, two reports were published in addition to Doré's. Subsequent to Doré's publication, 72 cases have been reported. Recent writers have referred rather loosely to "about 130 cases reported in the literature," but the total is nearer 242.

Not all summaries of case reports can be taken at their numerical value. For instance, Kohl[86] included in his list of lingual thyroids the case of Barwell[205], of Ogle[229], and two cases of Glas[212]. Barwell's was a lateral aberrant goitre, Ogle's was not lingual, and Glas' were adenomyomata, for which Glas made no claim of thyroid relationship.

Cattell[38] reported only two cases among 7,600 operations on the thyroid gland at the Lahey Clinic. Ulrich found only two

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among 4,000 at the University of Pennsylvania Hospital, and Urban's[179] was his first in 2,500 cases of thyroid disease.

In the past 20 years at the Wilkes-Barre General Hospital there have been 2,620 cases of thyroid disease. Of these, the case to be reported has been the only one of thyroid tumor of the tongue.

Lenzi[92] in 1918 called his the thirty-second case at a time when 162 can be culled from the literature. Strauss[167] in 1906 considered his the forty-third when 85 were on record, and in 1910, Livi[96] credited only 35. In 1907, Ungermann[178] regarded his the thirtieth case.

THE FIRST CASE.

The first case is credited by all writers to Hickman, an Englishman, who reported in 1869, the death by suffocation of a newly-born baby with an enormous thyroid tumor in the base of the tongue. A close study of the description by Hunt, an American, in 1865, of his experience with a tumor on the base of the tongue in a young woman, will suggest here was a case of thyroid tumor, although not proven. No illustrations are given and no histological description in either the article in the *American Journal of the Medical Sciences* or the duplicate article in the *Transactions* of the College of Physicians of Philadelphia, but his description might have been that of any one of the 200 proven cases that followed in the literature, and the postoperative course was almost identical with the experience Haynes had in 1912 with a young woman, from whom he had removed a proved thyroid tumor of the tongue. Hunt did not remove his growth, but left it *in situ* to slough away after strangling it with ligatures. It sloughed well enough, and the patient died. He was very emphatic that the death was not due to strangulation; in fact, William Pepper did the tracheotomy that was to no avail. The patient's dizziness, weakness, nausea and vomiting, and final collapse correspond startlingly well with the patient's experience in Haynes' case.

Hunt described a tumor in a female, age 16 years, situated on the posterior part of the tongue and immediately in front of the epiglottis. "It apparently completely filled the fauces, but the velum could be readily raised from it and the finger passed behind and around its posterior surface and upon

drawing the tongue forward and depressing it, the parts behind were seen to be entirely normal. In all other respects, the patient appeared to be perfectly healthy. Five years before (*i. e.*, before puberty), a physician had examined the throat and at that time the tumor was the size of a small pea on the middle of the posterior surface of the tongue. The surface of the tumor was very red, with large distended vessels coursing over it. There was no pulsation. It was quite elastic, and had an indistinct fluctuating character but not decided enough to indicate fluid. The opinions as to its nature were various but favored the view that it was an hypertrophy of glandular tissue and probably connected with the part known as the foramen cecum at the apex of the circumvallate papillae." After describing the operation, he says: "An incision was then made into it and a small amount of a peculiar gelatinous-looking substance exuded. But there was no disposition to empty itself. The material was at once examined under the microscope and its character agrees more with the adenoid or glandular variety of morbid growth than with any other form."

The operation was on Saturday. "On Sunday she had some sickness of stomach, with vomiting. By night, had marked fever, pulse running up to 120 — is very sensitive to light and wishes the room darkened. On Monday the same symptoms combined in a more aggravated form." (He attributed them to the mass of strangulated, sloughing tissue.) "One A. M. (Tuesday) she complained of lightness in the head. At 2:30 A. M., a sense of smothering." At 5 A. M. she was unconscious, respirations were limited, lips blue, face livid, the pulse small, quick and irregular. There was no relief from drawing the tongue forward, nor from tracheotomy, and she died four hours later.

Autopsy showed no edema of the glottis, the larynx and trachea showed no abnormality. As to the cause of death, Hunt thought it was not suffocation. He thought it was "transference of irritation from the ligated tumor to the respiratory centres and that the vessels of the brain became secondarily involved as indicated by the 'lightness in the head' and the drowsiness." The third supposition was "coincidence."

Now compare Haynes' experience in 1912 with a proven case of lingual thyroid which he had operated upon. This

was in a female, age 18 years, with a tumor at the base of the tongue, covered with dusky mucous membrane with a network of vessels. It was tense, elastic, not fluctuating, fixed and not painful. This was removed by the suprahyoid route and had a dense capsule as of a very vascular hypertrophic thyroid, although it was easily shelled out. "The postoperative course was very interesting." (She was operated upon Nov. 9.) "On Nov. 24 she had an attack of dizziness, falling off the chair apparently in a faint. She complained of pain in the back of the head and said she could not see. She was nauseated and vomited during the night. On the following day she felt weak and sick and could not leave her bed. She refused all food. On Nov. 26 her temperature was 101°; pulse, 100. She lay huddled up in bed with the covers drawn tightly around her, with her eyes closed in a 'dopy' state. She could be aroused and answered when spoken to, but volunteered no information and paid no attention to her surroundings. Otherwise the examination was negative; she was put on thyroid extract in tablet form and under this medication she steadily improved."

It would appear that Hunt was describing an experience with an unrecognized thyroid tumor of the tongue, and that had it been named so, he could be given the honor that has been Hickman's, of recording the first recognized case of thyroid tumor of the tongue.

THE DEVELOPMENT OF THE TONGUE.

The tongue consists of two portions, the body anteriorly and the base posteriorly. The anterior portion is formed from the region between the first and second branchial arches, from which project dorsally the *tuberculum impar* of His. In 1885, His held that the tongue was formed by enlargement of the tuberculum impar. Immediately behind this latter is a deep evagination of the epithelium (embryos of 5 mm.), which is the median thyroid evagination, and behind this again is a transverse elevation formed by the ventral ends of the second and third branchial arches, the *copula*. The part corresponding to the foramen caecum is the remains of the median thyroid evagination. According to Keibel and Mall (2:343), as early as 1869 Dursy had described the body of the tongue as having a paired origin, a condition more recent-

ly described as occurring in man by Kallius and by Hammar (1901). From the anterior portion of the *copula* and the neighboring portions of the second arch, the root of the tongue develops.

THE DEVELOPMENT OF THE THYROID GLAND.

The thyroid is one of the earliest recognizable structures developed in the embryo, being found in those 3–4 mm. long. The anlage appears almost contemporaneously with the formation of the first pharyngeal pouch or only a little later, as a prominence on the ventral wall of the pharynx. It soon becomes constricted to form a stalked vesicle, the hollow stalk being the *thyreoglossal duct* of His. The vesicle becomes a solid pyriform mass, which enlarges caudalward as a bilobed mass and becomes isolated from its origin at what later becomes the base of the tongue, by constriction of the upper end of the epithelial cord which is the obliterated thyreoglossal duct. The site of origin after birth is marked by the foramen caecum. The duct usually disappears, but may persist as a hollow epithelial cord extending from the tongue toward the thyroid body. Cysts forming along the line of this thyreoglossal duct may persist after birth, not to be confused with islands of thyroid tissue left along the same tract, although thyroid tissue has been identified in the walls of such cysts.

Bochdalek (1886) described such remains as a blind pouch opening on the base of the tongue and called it the *canalis excretorious linguae*. His (1885) called this canal the *ductus lingualis*, later (1891) referring to it as the *ductus thyreoglossus*, when he had occasion to remonstrate with Kanthack (1891) who had denied the findings of His in what appears to be a rather inconclusive, but controversial article. Said His: "Had Kanthack been acquainted with the work of Streckeisen, which he seems to have overlooked, so would he have been spared his doubt." A. Vater (1723), cited by Erdheim, had introduced a sound deeply into the foramen caecum and believed this to extend to the thyroid gland and thus act as an excretory duct for the thyroid.

The isolated bilobed mass of embryonal thyroid tissue as it descends in the neck, is composed of irregular cords of cells, disposed for the most part transversely, and enlarges laterally

and dorsally. The lateral lobes are united by the isthmus which represents the main body of the median anlage and may extend upward as the pyramidal lobe. The lateral masses engulf the more caudal portion of the outpouching from the fourth pharyngeal pouch, the so-called ultimobranchial (postbranchial) body, but this contribution in man undergoes reticulation and degeneration and disappears (Kingsbury, 1935). Verdun, cited by Keibel and Mall, had previously rejected the name *lateral thyroid anlage*, which had been applied to the ultimobranchial bodies by the older anatomists. Badertscher (1918) had found in the pig that the ultimobranchial bodies participate only to a small degree in the formation of thyroid follicles. In thyreoaplasia, the defect of (middle) thyroid anlage, the ultimobranchial body gives rise to no thyroid tissue (Maresch, 1898; Puecker, 1899; Erdheim, 1904; Dieterle, 1906). Lahey (1924) argued that development of myxedema following the removal of lingual goitre clinically refutes the possibility of thyroid tissue arising from the ultimobranchial bodies. If a lingual goitre represents only the thyroid tissue arising from a median anlage, then, even though this be removed, myxedema should not result if lateral anlagen exist because they should have developed thyroid tissue which would have prevented it.

Accessory thyroid bodies after birth occur somewhere in the tract along which the median anlage has descended during its ontogeny, but a second group may arise in a somewhat different way. The thyroid enlarges by lateral sprouts, and apparently one or more of these may become detached from the rest of the gland. The shifting backward, in a relative sense, which subsequently occurs in connection with the main mass, may leave these detached bodies behind, and as the third pair of aortic arches straighten out to form the internal carotids, they may become closely associated with them. In this way one may explain the occurrence of isolated masses of thyroid tissue in the region of the upper part of the neck, and in the neighborhood of the angle of the jaw, which have become enlarged and subsequently removed (Peter Thompson, 1910).

Cattell (1931), however, citing Wenglowski (1912), seems to lean to the view of the older anatomists, saying that groups of cells arising from the ultimobranchial body, pass downward and come into relation with the main thyroid body. He

cites Norris (1918) who showed that these cell groups fuse with the lateral lobes of the thyroid, but atrophy and form no part of the normal thyroid gland. He believes that an arrest in the lateral descent will account for lateral aberrant thyroids, and that this is a logical explanation of their origin.

Gachet's[52] case of left-sided lingual goitre, with a normal pretracheal right lobe, was an interesting example of associated maldevelopment. This man had also hemiatrophy or maldevelopment of the left mandible and lack of development of the left internal ear.

ABERRANT THYROID TISSUE.

Eiselberg, as well as many other authors, gives credit to Verneuil for first recording the discovery of thyroid gland tissue isolated elsewhere in the neck from the main thyroid body. Verneuil himself, however, (1853) gives prior credit to Cruveilhier (1852) for recognizing thyroid tissue elsewhere in the neck than the normal location, and to Le Gendre (1852) for prior description of the pyramid, along which they found thyroid rests. Says Verneuil: "I have little to add to this description (Cruveilhier's). In later times, in dissecting the insertion of the muscles of the tongue to the hyoid bone, I have run across a small mass, of glandular appearance, strongly adherent to the lower part of the superior border of that bone, between the geniohyoid and the genioglossus. This mass was red, soft, buried, of the volume of a large pea, with a smooth surface, of a homogeneous tissue; I have examined it microscopically and found there thyroid gland tissue."

The Italian writers trace evidence of reports of isolated thyroid gland tissue to earlier writers. Notably Cecca (1901) says: "From the observations of Morgagni, Laluette and Santorini, it was apparent in the eighteenth century that some of the observers suspected the possible presence of accessory thyroids in the suprahyoid region. Albert Haller (1756) recognized these isolated glandular nodules which he mentioned but did not describe, saying: 'I have seen a real gland that was situated before the median thyroid cartilage but did not belong to the thyroid itself'." Fiori (1903) gives priority to Albert von Haller (1779) for the description of aberrant thyroid nodules. Gruber (1876) also gives priority to Haller

by an earlier date *(Elementa Physiol.*, Tome 3, p. 196, 1766) for recognition of a separate gland springing from the cornu medium. According to Cecca, Luigi Porta (1849) devotes an entire page to the description of two or three small nodules separated from the thyroid but connected with it through a pedicle and having the same texture, and seen both in children and adults. "Porta's observations escaped the attention of foreign savants so that Bruch in 1852 and Verneuil in 1855 reported several examples of accessory thyroid glands as something new." "Virchow in 1863 confirmed Verneuil's observation that in two cases the pyramid had lost its connection with the main part of the gland to which it was united with a long thin filament, from which he assumes that the pyramid may itself simulate accessory lobules if you are not careful in dissection. But Morgagni made the same observation 150 years before."

Gruber (1876) made a study of 300 Russian cadavers, confirming completely the work of Porta, but adding several important points: *1.* He was the first to call these lobules "accessory thyroid glands." *2.* Described accessory lateral and inferior glands. *3.* Admitted the existence of accessory glands far away from the thyroid, and although he never questioned the common origin, he was never able to give explanation of it.

Zuckerkandl (1879) in 200 cadavers found in 57 instances a prehyoid gland, and in 14, one below the hyoid bone, but he did not consider the glands above the hyoid bone as accessory thyroids, inasmuch as he entitled his thesis, thus: "On a Gland in the Suprahyoid Region Which So Far Has Not Been Described." Zuckerkandl's observations were almost simultaneous with Kadyi (1879) who identified accessory thyroids and distinguished them as "Zungenbeindrusen" *(glandulae hyoides)*, dividing them into *glandulae suprahyoides* and *glandulae praehyoides*, according to their relation to the hyoid bone. He recognized these as rests of the middle lobe separated from the lateral lobes in the descent.

Wolfler (1880) substantiated these findings and explained their position before the hyoid bone through the influence which the development of the neck organs, especially the larger vessels, exerts on the epithelial rests, in which they press the same ventrally so that the union of the copula of the hyoid bone and the horns goes dorsalward.

Streckeisen (1886) felt that one has to differentiate sharply between the thyroid gland tissue and cysts of the hyoid bone, as heterologous structures. "The thyroid gland of the hyoid bone is connected with the processus pyramidalis: The cysts of this bone on the other hand possess intricate relation with the cysts of the root of the tongue and to these on the tip of the processus pyramidalis. Both, however, thyroid gland and cysts, are not distinct, but often combine one with the other."

Lahey (1924) identifies aberrant thyroids according to location as follows: Those which remain at the foramen caecum are the true lingual goitres; those within the tongue, the intralingual goitres; those below the tongue, the sublingual goitres; those in front of the larynx, the prelaryngeal goitres. Two other locations are within the superior mediastinum, and those just outside the sternomastoid muscle in the posterior triangle of the neck. "Allied accessory thyroid" is an extruded adenoma still attached to the thyroid by fibrous bands. "Pseudoaccessory thyroid" has an isthmus of true thyroid tissue, joining the mass to the thyroid. "True accessory thyroid" has no connection with the normal gland.

Other authors call the "accessory thyroid" that mass of glandular tissue existing at a distance from the normally situated gland. The "aberrant thyroid" is the entire mass of thyroid developing apart from the normal location at which no thyroid has developed. Lingual thyroid or goitre can then be either accessory or aberrant, depending on whether thyroid tissue simultaneously exists in the normal location or not. As will be seen in the analysis of the recorded cases of lingual thyroid, instances of both will be found.

The present discussion has only to do with lingual, intralingual and sublingual thyroids or goitres, the sublingual being the suprahyoid (Kadyi) and praehyoid (Kadyi, Streckeisen and Zuckerkandl).

ETIOLOGY.

Lingual thyroid is essentially an embroyological malformation, a vestigial rest appearing on the base of or within the tongue, or below it along the course of the thyroglossal duct of His. We can surmise no cause for this malformation. It was found in women in 74 per cent of the cases reported (78 in the female, 27 in the male, the sex not being indicated

in 37 cases). Because disturbances of the thyroid gland are more apparent in the female in puberty and by reason of pregnancy, it might be argued that as many cases occur unnoticed in the male, but escape attention because the gland is less active in the sexual sphere. Anglesio felt that the cases seen and reported are a very small percentage of the actual cases that must exist.

Of 140 reports in which such details were noted, 30 per cent occurred in females in puberty; 55 per cent occurred in females between ages of 18 and 40 years; 10 per cent in the years of the menopause, and 5 per cent in old age. Seven cases were noted in infants, in addition to those found at autopsy, which had not given evidence in life; and 11 in childhood. In Demuth's case,[38] a male, the growth first appeared in puberty.

Anglesio,[3] Carnelli,[30] Droesbecque,[42] Leulier,[93] Seelye,[149] and Rubeli[141] reported cases that had hemorrhage from the mouth coincident with menstruation, or in which the growth appeared coincident with the first menstruation, or fluctuated with menstruation. In Rubeli's case, there was alarming enlargement of a lingual thyroid in the last month of pregnancy, necessitating Cesarean section. The growth subsided promptly after delivery of the child. Rubeli regarded lingual thyroid an indication for interruption of labor, and a contraindication to breast feeding. Warren[188] reported a case in which the growth first appeared following the birth of her youngest child 30 years before. The compensatory development of a lingual thyroid following thyroidectomy was reported by van Selms[181] and following pathological destruction of the normal thyroid gland, by Willis[192].

Trauma appeared to be the inciting factor in cases of Goris,[61] Kohl,[86] and Walther.[187] Haynes,[72] Bloch,[22] Straus,[168] and Warren[188] reported the first appearance to follow a "cold," influenza, or upper respiratory infection. Wolf's case[194] appeared to date from whooping cough, Perkins'[127] from diphtheria, Anglesio's[3] from an intestinal infection with recurrences, Lenzi's[92] from typhoid fever, and Hartley's[69] from "fever." Caderas' case,[28] a male, dated his onset from exposure to war gas, and Zehner's[196] to the loss of her husband in war, a fear neurosis. "That such a developmental possibility can be assumed, is easily understandable in the variable dispo-

sition of women which stands in relation with the sexual sphere."

Hickman's case[75] caused suffocation at birth. The description of Meixner's case[110] has a dramatic appeal. A woman called to her maid and said that a fetus had been passed The navel cord was pulled off. The dead body was found under the couch, wrapped in a cloth, and attempts at resuscitation were fruitless. In the lung secretions and mouth were microscopic particles (dishwater elements). The thyroid in the neck was absent, but at the base of the tongue was a growth — pushing down on the larynx to close it. The opinion was that the child was born a few weeks before normal time, but could have lived only a short time anyhow because of the obstructive lingual growth, and that an unnecessary attempt had been made to drown it in dishwater.

APPEARANCES.

The typical lingual thyroid occupies the median line of the base of the tongue between the foramen caecum and the epiglottis, as a rounded, hemispherical mass, often lobulated but covered with normal intact mucosa, through which a varying degree of vascularity is marked by a plexus of superficial veins. The color is usually reddish, of a different hue from the tongue itself, but varies to bluish. It is circumscribed, distinct from the glossal structure, on a broad base, and not attached to the lateral or posterior confines of the fauces, reaching to, but not adherent to the epiglottis which may be pressed backward to such a degree as to cause suffocation. When visible, it may reach such size as to "fill the pharynx" but, as will be seen, may be submerged in the glossal musculature or may be only small islands within the base of the tongue. The cases reported to have had a pedicle are limited: Livi,[96] Harvey,[70] Moulonguet,[119] Caderas[25] and Bishop.[18]

The sublingual variety may present itself in the submental region as a rounded swelling beneath the skin, fixed to the musculature but circumscribed, and moving with the movements of deglutition.

The lingual tumor is semisoft or firm, elastic, not fluctuating, painless and not tender, not blanching with pressure. The

palpating finger can be passed around it, and between it and the epiglottis.

SYMPTOMS.

The subjective symptoms are those of a mass or foreign body in the throat, sufficient to give a constant desire to swallow something that will not be moved. Dysphagia is a common complaint, and yet with very large tumors of this sort, there has been no difficulty in swallowing even solid food. In the face of the common complaint of "something stuck in the throat," lingual thyroid should be borne in mind and ruled out.

Dysphonia, the "pharyngolalia" of the Italian writers, is common. Dyspnea, especially nocturnal, is usual and has been described of alarming proportions obtaining the degree of suffocation. One case received "energetic cauterizations of the nasal turbinates" for obstructive breathing, without being recognized as a lingual thyroid tumor.

The abundant vascularization of the thyroid gland in this exposed location, has led to hemorrhage from the mouth, varying from blood-streaked saliva to abundant and alarming hemorrhage. Indeed, this has been a common initial symptom. When hemorrhage has occurred, it is usual to find eroded areas on the surface of the growth.

Despite the apparent possibility of frank symptoms developing from a growth in this location, cases are recorded in which the subject has reached death in old age without any known complaint referable to this site. Many others have been recognized casually by the patient, a friend, or an examining physician, without any symptom before the discovery having suggested such a growth, and others having been recognized, have produced no symptoms or complaints which have demanded removal of the tumor.

Endocrinal disturbances in connection with lingual thyroid are usually not due to the lingual tumor itself. It happens, however, that a lingual thyroid may be the only thyroid tissue present, and by the degenerative processes common to the thyroid gland, produce disturbance of the endocrine system which may then be properly attributed to the lingual tumor itself.

PATHOLOGY.

Lingual thyroid is essentially a benign tumor, but may, by grave hemorrhage, alarming dyspnea, or constant dysphagia, be malignant in its effect. Histologically, the cases of malignant degeneration are few. It is common for the lateral aberrant thyroids to undergo malignant change, but the lingual thyroid, almost never. Gunn's case[62] (1910) was diagnosed "carcinomatous thyroid tissue." There was no description of the sections, but he presented "an exquisitely made model." Ashhurst and White[6] recorded a case in 1925 as the first of carcinoma in an aberrant thyroid at the base of the tongue. Moulonguet[119] in 1930 recorded his as to him the first authentic case of cancer (adenothyroidome). Rutgers[143] in 1910 had recorded an anatopathologic diagnosis of malignant adenoma. Brentano[24] in 1911 had recorded his case in which nine years before a growth had been removed from the left half of the tongue, to be followed in two years by removal of glands from the right submaxillary region. Shortly thereafter there was recurrence in the tongue and involvement of the glands of the left side. He regarded these growths as metastatic and the lingual tumor as malignant. Thyroid was present in the normal pretracheal site. Finally, Levi and Hankins[94] in 1935 have recorded a case showing "low-grade carcinoma of thyroid tissue," in which the normal thyroid gland was not palpable in the neck, and in which myxedema followed extirpation of the lingual tumor.

The relationship of lingual thyroid to myxedema and cretinism is interesting. Strada[165] says that Peuker's was the only case of congenital myxedema with absence of the normal thyroid gland, in which thyroid tissue was not found in the tongue. Dieterle's,[40] however, should be included here. Several of the case reports described the pathological study of cretins at autopsy. Such were those of Aschoff,[5] MacCallum and Fabyan[100] and five cases of Erdheim.[44] Here death occurred early in life from athyreosis, and the only thyroid tissue was that found in the base of the tongue, rudimentary and insufficient.

Adult cretins were described by de Boncourt,[23] Brunner,[25] Didier,[39] Harvey,[70] Krassnig,[88] Lindt,[95] Leulier,[93] Matti,[104] and Rebattu[135]. In none of these were thyroid glands discoverable in the normal location, and the lingual thyroids were

insufficient. Where it was necessary to remove the latter, it was done because of hemorrhage or extreme enlargement giving severe dyspnea or dysphagia.

Improvement in thyroid function, on the other hand, has followed removal of a degenerated lingual thyroid. Such was the case of Roegholt[128], which had never menstruated until after operation. Urban removed the lingual growth and implanted thyroid gland tissue. He thinks the postoperative improvement was due more to the lingual thyroidectomy than to the implanted glands. Rabinowitz[134] did a partial extirpation of a lingual thyroid adenoma with resultant improvement in the general condition. Krassnig[83] practiced autoimplantation from the lingual tumor. Austoni's case[7] improved after operation.

Postoperative myxedema has followed in many cases the extirpation of a lingual thyroid. Goris,[60] Lenzi,[92] Lindt,[95] Meixner,[110] Reintjes,[136] Seldowitsch,[150] Shurly,[152] Ziegelman,[197] reported instances of this sort.

On the other hand, Didier[39] reported improvement in the preoperative myxedema. Lindt[96] says that one should not hesitate to remove a tumor giving symptoms, inasmuch as the danger of myxedema is not so great in the present day with modern methods of treatment. Ulrich[177] advises exploration of the neck to determine the presence or absence of thyroid tissue in the normal location and describes two cases in which it was absent and in which the lingual tumors as a consequence were not disturbed.

Asch[4] reported the first case of postoperative tetany, in which he was not certain that thyroid tissue was present in the neck. The sections showed epithelial bodies (parathyroid). Two years later, Wood[195] demonstrated parathyroid tissue in two specimens of lingual goitre. A third specimen not only showed thyroid tissue, but also remnants of the thyroglossal duct bearing ciliated epithelium (thus refuting Kanthack). Dieterle[40] had demonstrated epithelial bodies in the base of the tongue in his case of congenital athyreosis.

DIFFERENTIAL DIAGNOSIS.

Lingual thyroid tumor is to be differentiated from the following benign tumors which may appear at the same site:

Angioma: This is purplish, soft and compressible, blanching under pressure and refilling on release.

Amyloid: The only case so reported in the appended table was Hanzel's. It probably represents an advanced stage of degeneration of a thyroid nodule, and its differential diagnosis is not of practical interest.

Adenoma: This is firm and embedded and resembles the thyroid tumor. Several of the cases reported have been classed as adenomata.

Cysts: Cysts of the thyreoglossal duct are difficult to differentiate from lingual goitre. Ordinarily the contained fluid imparts a fluctuant sensation to the finger, as opposed to the resistant and elastic quality of the thyroid tumor. The presence of superficial veins and the absence of the isthmus in the neck suggest thyroid tumor. Aspiration of the contents should show clear viscid material from a cyst, and may produce collapse of the walls. The cyst is usually lighter in color and may be translucent. Mucous cysts or ranulae are not typically in the base, but beneath the tongue anteriorly or laterally.

Dermoid cysts and tumors are classed by some writers with thyroid tumors of the tongue, having a common origin. The differential diagnosis may not be possible before removal.

Chondroma is rare and this study showed no case of osteoma. The former is more firm and lacks the lobulation of the thyroid tumor.

Fibroma: This is fairly frequent, usually in the male, smooth, hard, more often pedunculated without the varicosities of the thyroid tumor.

Gummata: These are usually multiple, often ulcerated, associated with adenopathy. The diagnosis should be clear by the serologic and therapeutic tests.

Hypertrophies of the Lingual Tonsil: These are flat, verrucose, superficially located and distributed anterior to the foramen caecum. They are usually multiple and separated, but may be confluent and raised, without the vascularization of the thyroid tumor.

Lipoma: This is rare, soft, yellowish, lacking the firm and elastic quality of the thyroid tumor. By preference it occupies the anterior portion of the tongue, is rounded and circumscribed.

Lymphangioma, like angioma, is soft and compressible. It is pale and may be translucent.

Papilloma may be sessile, but is more usually pedunculated. It is reddish to pale, soft, and other locations are favored in the fauces than the base of the tongue.

Of the malignant tumors, epithelioma and lymphosarcoma are to be differentiated. Carcinoma of the tongue usually involves the anterior half or the tip along the margins. It may appear at the base, but usually to one side at the base of the tonsil. It is indurated and infiltrating. In any situation where thyroid tumor is suspected, but not proved, biopsy is indicated by aspiration or excision. In many of the cases reported in later years, as will be seen in the appended table, especially where the tumor was not excised for fear of myxedema, its nature was proved by histological examination of an excised portion.

TREATMENT.

There is no doubt that not all lingual thyroid tumors call for active treatment. Certainly, there is a note running through the more recent writings advising conservatism. Many of the tumors appearing at puberty must represent physiological hypertrophy and might better be handled as are analogous hypertrophies in the normally situated glands. Many represent the only functioning thyroid tissue present. Rest and symptomatic treatment with the administration of iodine and thyroid extract are more advisable than too hasty surgical intervention. If the latter must be done, the presence or absence of the normally situated gland should be determined. In this connection one may reasonably doubt the statements of those who have claimed to have proved its absence by the classical tracheotomy incision. Unless the tissues lying laterally are dissected, the classical tracheotomy incision will prove only the presence or absence of the isthmus. Those who have advocated dissection of the pretracheal region by the collar incision have a more substantial ground for their argument.

Marked dyspnea, dysphagia and dysphonia, and recurrent or grave hemorrhage, may well demand surgical intervention. More recently electrocoagulation has been used with successful shrinking. Many have been removed simply by the direct transbuccal route, by the cautery, snare, or incision and dissection. Goris[60] and Luks[98] employed the sublingual approach through the mouth. Stuart-Low[169] split the tongue from tip to base. Monro and Taylor[118] and Brunner[25] split the cheek.

Of the external operations, those by the hyoid route have been most employed. Lateral pharyngotomy was employed by four operators, and four others required temporary section of the jaw. The hyoid route is either suprahyoid or subhyoid, by a vertical median incision or by a transverse incision. Division of the hyoid bone is permissible. Matti[105] discusses in detail the several techniques. Pauchet[226] gives an excellent and well illustrated description.

The question of tracheotomy has been given much more debate than would seem necessary. Circumstances must determine whether or not the trachea should be opened. The collar incision is more certain for exploring the cervical thyroid gland, and the exploratory vertical incision is no argument for routine tracheotomy. If it is not done in preparation for lingual thyroidectomy, provision should be at hand for opening the trachea in the event of dyspnea or alarming hemorrhage during the operation.

REPORT OF CASE.

N. W., female, age 14 years, for a year had observed a growth at the base of the tongue, not increasing in size during that time. More recently it had caused irritation, moderate dysphagia, and had induced the feeling of a necessity to swallow something lodged in the throat. There had been no pain and no bleeding. Her general health was good.

She was undersized, although the general nutrition was good. She was rather pale, but the skin and hair of good texture. General examination was negative. The teeth showed defective calcification, the mouth and tongue were clean. At the base of the tongue, in the center at the region of the foramen caecum, was a rounded growth, elevated about 0.5 cm., about 1.5 cm. wide and $\frac{3}{4}$ cm. long. It was visible without

depressing the tongue. It was smooth anteriorly, covered with normal mucosa, through which several superficial veins coursed, of a light, dull, reddish-brown color. The edges were rounded from a broad base and it was attached to no other surrounding structures. It was semisolid; not painful.

The lateral lobes of the thyroid gland were palpable in the normal location in the neck, but the isthmus was lacking. The trachea was in midline and palpable through the skin. X-ray examination of the pharyngeal region was negative.

The diagnosis was aberrant thyroid gland at the base of the tongue.

Operation was on March 17, 1933, under ether anesthesia following ethyl chloride induction. The head was low and no tracheotomy was done. The tongue was fixed with two stay sutures, on either side well back, and by these drawn forward. This presented the growth well forward as a lobulated circumscribed, reddish tumor at the foramen caecum. It was fixed with forceps and excised by wedge-shaped incisions pointing toward the midline of the tongue. There was no excessive bleeding. The defect was closed by three interrupted silkworm gut sutures in the median line, and the stay sutures removed. The silkworm gut sutures were removed on the fourth day and the defect healed by first intention. She was discharged on the fourth day.

Microscopic Examination: R. R. Janjigian, M.D., pathologist: The section presents a complexity of tissue. It is enclosed by a basement membrane and the epithelium is flattened. Within appear two different types of acini, filled with colloid material suggesting thyroid gland. The vesicles are lined with epithelium and show marked hyperplasia. The glands are tortuous and enlarged and colloid hypertrophy also is obvious. (Cyst?) There are also remains of mucous glands in different parts of the section, suggesting mucous glands characteristic of lingual glands. Diagnosis: Lingual thyroid.

Postoperative Examination: She was examined again on Oct. 22, 1935, at which time she was 16 years old. Her mother reported her sluggish in physical and mental activity, tiring easily, although she answered questions alertly and was in first-year high school. She was said to be "cold all the time," especially in winter and especially in the extremities. She had

not yet menstruated, although her grandmother, mother and sister had not themselves menstruated until the age of 17 years. She had a cretinoid appearance with sallow skin, but no pseudoedema, and her cephalic hair was of good texture and quality. The breasts were not developed and the pubic hair was scanty. She weighed 70 pounds and was four feet, six inches tall (42 pounds underweight and eight inches undersized). The basal metabolic rate was +2.5. Her voice was flat and low. There was no evidence of the growth at the base of the tongue, which was smooth, but the lateral lobes were enlarged.

CONCLUSIONS.

1. Lingual thyroid tumor is a developmental abnormality presenting at the base of the tongue, or within or beneath the tongue, resulting from a vestigial rest of the thyroid anlage.

2. The precaution should be made of ascertaining the presence or absence of other thyroid tissue in the normal location before removing the lingual growth for the relief of symptoms.

3. The lingual tumor may produce grave dyspnea, dysphagia or hemorrhage, but is unlikely to undergo malignant change.

4. It usually appears in the female, at the several periods of endocrinal hyperactivity: Puberty, pregnancy, and the menopause.

5. Removal of the lingual thyroid may be, and often has been, followed by myxedema. The postoperative appearance of tetany has been reported and points to the association in development between the thyroid gland and the parathyroid.

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(To be continued in November issue.)

TUBERCULOSIS ASSOCIATED WITH ESOPHAGEAL OBSTRUCTION.

DR. HARRIS P. MOSHER, Boston.

The purpose of this paper is to report three cases of tuberculosis, two being of the chest and one of the abdomen, all three associated with obstruction at the terminal portion of the esophagus. Also, I wish to put on record the autopsy findings in the esophagus of a patient who died of pulmonary tuberculosis. The central portion of this esophagus for some five inches showed a moderate spindle-shaped dilatation with marked thinning of the esophageal wall. Above and below the thin portion the musculature was of normal thickness. Small glands about the size of half a pea (3–4 mm.) were found at the top, the bottom and the middle of the specimen adherent to the esophageal wall. These showed giant cells and tubercles. Vertical rougae were present at the top and at the bottom of the specimen where the thickness of the esophageal wall was normal. They were absent over the thin portion. The surface of this was perfectly smooth (see Figs. 1, 2, 3 and 4).

The three cases will be reported and discussed in the order just given.

CONCLUSIONS.

In the three cases reported the obstruction at the terminal portion of the esophagus reacted differently to dilatation from the usual case of fibrosis due to other forms of infection. In the first case the constriction dilated easily to normal, yet the patient after two years still has to feed herself by tube. In the second case, little or no dilatation was accomplished. The third case, in whom only mercury bougies were used, clinically regained normal swallowing by the constant use of the bougie; yet after nearly a year the fluid level is still at the arch of the aorta. In the first case dilatation precipitated a mediastinitis and a tubercular pleurisy. In the second case

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the withdrawal of a large amount of fluid from the abdomen greatly relieved the obstruction to swallowing. The writer feels that the toxins of chest tuberculosis can cause a localized atrophy of the musculature of the esophagus leading to a

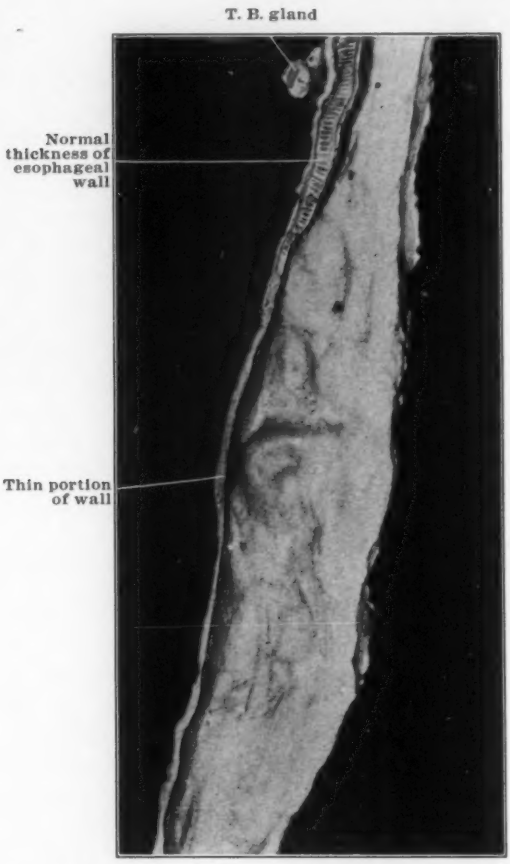


Fig. 1. Spindle shaped dilatation of esophagus. Case of pulmonary tuberculosis. A35-91-4.

spindle-shaped dilatation. This can occur without fibrosis of the wall of the esophagus or tubercular infiltration. If there is such a disease as idiopathic dilatation of the esophagus about which at one time there was much discussion, its path-

ology could be explained by the findings in this autopsy specimen, namely, a thinning of both muscular layers in the dilated part of the esophagus, the thinness and looseness of the circular layer being especially striking.

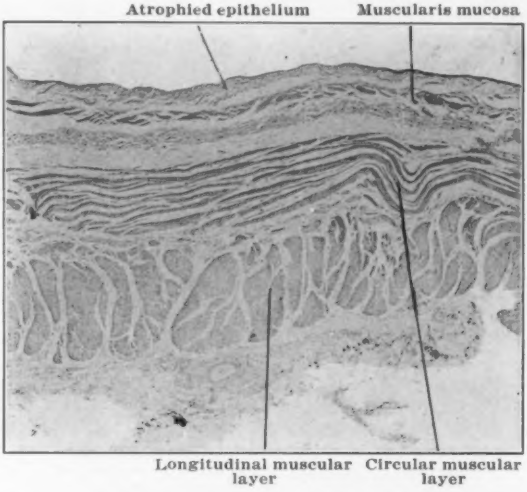


Fig. 2. Spindle shaped dilatation of the middle third of the esophagus. The epithelium has disappeared. The muscularis mucosa and the circular muscle are very much atrophied. Auerbach's plexus is nearly absent. The section is from the dilated portion of the esophagus. Case of pulmonary tuberculosis.

K. S., age 37 years. F. White. Native. Graduate nurse. H. C. No. 336200. Admitted to M. G. H. April 1, 1934. Service of Dr. E. D. Churchill. Discharged April 19, 1934. *Admission Diagnosis:* Esophageal stricture; mediastinitis. *Final Diagnosis:* Pleurisy, serofibrinous; stricture of the esophagus. *Past History:* Negative up to five years ago. *Family History:* Negative. *Chief Complaint:* Chills and fever 10 days.

The patient was well and had no symptoms bearing on the present illness until five years ago when she entered St. Luke's Hospital in New Bedford with a ruptured appendix. She was in the hospital for two weeks.

The first three or four days it was necessary to pass a large tube for gastric lavage every four hours. No complications were noted at the time.

Seven months later, Nov., 1930, she gradually developed dysphagia and was informed she had an esophageal stricture. This time she weighed 148 pounds; slightly over one year ago she had especial difficulty in eating, so much so that her weight fell to 102 pounds. In other words, she lost 46 pounds (see Fig. 5).

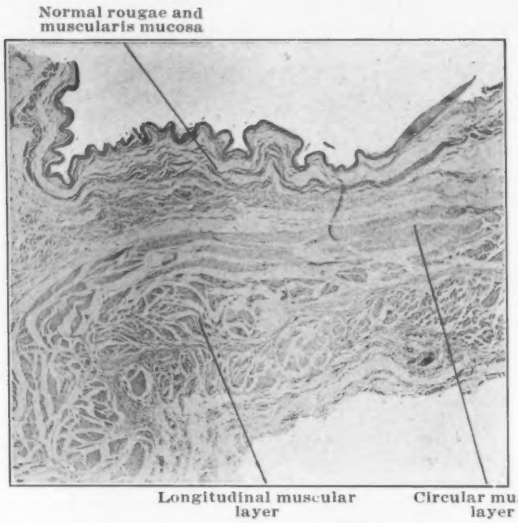


Fig. 3. Spindle shaped dilatation of the esophagus. This section is taken above the dilatation where the esophagus is of normal thickness. In this portion of the esophagus normal rougae are present.

In May, 1933, about one year ago, the patient was seen by Dr. Mosher who has dilated her esophagus, the periods varying from one week to one month up to the present time. She, herself, has passed bougies, size No. 32 French, whenever she felt the need.

During the past two and one-half years, the patient has occasionally complained of "substernal knots of pain" which she felt were constrictive, and at the same time she felt as though peristalsis was stopped in her intestines. When the peristalsis began again the pain in the chest went away, this pain averaging four days in duration and was severe only at the onset.

Six weeks ago the patient developed a dry pleurisy in the right chest with pain chiefly in the right axilla. The pain lasted about a week but her temperature, which averaged 102°, persisted for only the first three days. An X-ray of her chest at that time was negative.

On Feb. 6 the patient saw Dr. Mosher. On account of the recent pleurisy no dilatation with the bag was carried out; fluoroscopic examination only was made.

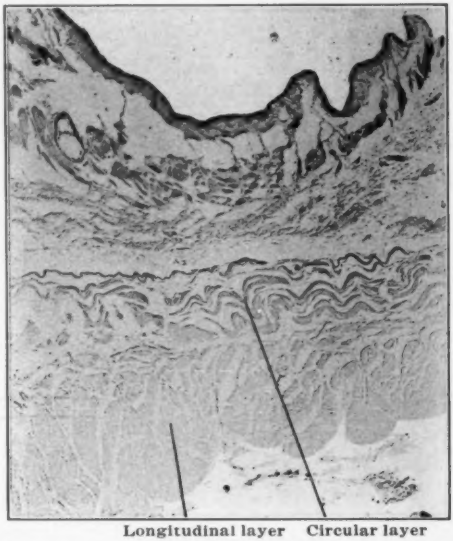


Fig. 4. Spindle shaped dilatation of the middle portion of the esophagus. Case of pulmonary tuberculosis. Notice atrophy of the circular muscular layer. The epithelium has disappeared. The muscularis mucosa is present but atrophied in part. Above this, the specimen is broken.

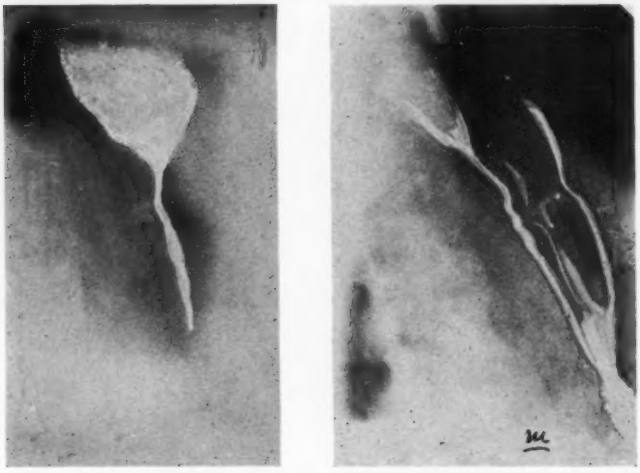
Twenty-five days ago she experienced one of her "substernal knots of pain" as above, radiating to both sides.

Twelve days ago she experienced another knot of pain, substernal, which lasted one day.

Ten days ago her esophagus was again dilated by Dr. Mosher without anesthesia. She may have had a slight temperature before this was done. Unfortunately, her temperature was not taken. Dilatation was done at 1 P. M. and at 3 P. M. on the same day she started to develop chills, fever,

with some pain in the right chest, behind and below; however, the next day she was able to work for two hours or so and she passed a bougie on herself.

Seven days ago her temperature was 103°. Her chest plate showed a thickened interlobar septum on the right with the right costophrenic angle obscured as if by fluid. At this time she was being cared for by Dr. Black at the Belmont Hospital, Worcester.



A B

Fig. 5. Miss S. The first figure (A) is a retouched X-ray film showing that the esophagus ends in an awl-like point, above which it is moderately dilated. This awl-like point is typical of fibrosis of the terminal portion of the esophagus (cardiospasm). In spite of the fact that the barium X-ray shows that the esophagus is practically closed, a No. 30 F. flexible finder can be passed in the majority of cases. In those cases in which the finder passes but the diagnostic bag will not, it is necessary to dilate the constriction with graduated bougies fitted with the flexible finder. This is done under fluoroscopic control. After two years the esophagus in this patient shows the same picture (B) except that the dilatation has increased. The second figure is a retouched X-ray film with the diagnostic bag in place. It inflated with 2 lb. pressure to normal width, that is, to 1 in.

Five days ago another chest plate showed that the entire right side was hazy. Physical examination placed the fluid as high as the fifth rib.

Three days ago, and seven days after the onset of the chills and fever, another bougie was passed at the Belmont Hospital. The chest was tapped and 900 cc. of clear straw-colored fluid were withdrawn from the right side. The specific

gravity was 1.016. The differential count showed polymorphonuclear leukocytes predominating with some lymphocytes and monocytes. The culture was negative.

Saturday, two days ago, a gastric lavage and feeding was given.

During this time she had no other change in her symptoms except for the expectoration of much mucous from her larynx, occasionally leading to vomiting for the past 10 days.

This summarizes the patient's history up to her transfer to the Massachusetts General Hospital, April 1.

Progress Notes at M. G. H.: April 2, the fluid in the chest on withdrawal was clear and straw-colored: there were 52 cells per cmm.; 90 per cent polymorphonuclears; no acid fast bacillus seen. No organisms with Gram stain. April 3, vomitus negative. April 5, the temperature continues high. There is marked deafness today. Ears checked by Dr. Mosher who advises waiting. Both middle ears showed serous exudate. The deafness cleared up in a few days without treatment. Hinton was negative. April 10, patient seen by Dr. Donald King who feels the diagnosis is acute tubercular pleurisy. April 18, patient seen by Dr. Lord. His opinion was: Neighborhood infection from mediastinum. Without more definite evidence of mediastinitis, do not advise operative interference.

Dr. Churchill's final opinion was: There is a question of localized mediastinal or periesophageal abscess with neighboring fluid in right pleura.

A series of X-ray plates were taken during the patient's hospital stay. The most informing one is as follows: Pleurisy on the right side with partial encapsulation. Collapse of the right lung. No definite X-ray evidence of mediastinitis.

April 18, chest tapped; 15 cells per cmm. Differential count: polymorphonuclear leukocytes, 6; lymphocytes, 93; mononuclears, 1.

At her entrance to the hospital a guinea pig was inoculated with fluid from the chest. After the usual period the pig was killed and was reported as positive for tuberculosis.

Naturally, what Dr. Churchill and I were watching for was a mediastinal abscess, hoping that it would localize in a place where it could be reached surgically. I expected to find it at

the lower end of the esophagus. Fortunately, it never materialized. April 19, the patient discharged.

Miss K. S. reported a short time ago for a check-up, Feb. 29, 1936. It is now two years since her tubercular pleurisy following dilatation of the esophagus. She still finds it necessary to pass a mercury bougie and to feed herself by tube. This is not the usual history of fibrosis of the terminal portion of the esophagus due to neighborhood infection. Dr. Black, the resident physician at the hospital where Miss S. worked, who has been responsible for her general medical care since the beginning of her trouble, devised an ingenious method of inserting a feeding tube at the same time the bougie is passed. The bougie is inserted as a staff inside a thin rubber tube the size and length of the bougie. The tube is fitted with a funnel at the top and has perforations at the lower end. The bougie and the tube are carried down together into the stomach. The bougie is withdrawn, leaving the feeding tube in place. This device has proved very useful because the patient still has to feed herself by tube.

Miss S. was extremely despondent at the beginning of her treatment. This struck me very forcefully because usually tubercular patients are so optimistic.

At Miss S.'s last visit, the X-ray plates showed arrested tuberculosis of the left apex. The lower end of the esophagus was markedly dilated and the crural canal was reduced to a diameter on the plate of about 2 mm.

During her dilatations and before her near tragedy the esophagus dilated readily with the bag. Light pressure of not over two pounds brought it up to normal and even beyond. At esophagoscopy, the esophagus showed a projection of the right crus into the field of the esophagoscope. This is not common. Usually it is the left crus which projects into the field of the tube. This same notching of the esophagus was seen in the X-ray film.

There is an adhesion of the diaphragm on the right and one is tempted to draw the conclusion that old infection at this point is the cause of the constriction on the right wall of the esophagus.

Sister M. C., W. F., age 29 years. Admitted Dec. 20, 1935. Discharged Jan. 28, 1936. Stay in hospital, 39 days. *Diagnosis:* Stricture lower end of esophagus. *Operation:* Esopha-

goscopy; dilatation. Readmitted Feb. 18, 1936. Discharged Feb. 29. Stay in hospital, 11 days. *Diagnosis:* Stricture of the esophagus. *Complications:* Tubercular peritonitis and colitis. *Chief Complaint:* Difficulty in swallowing. *Past History:* Measles. No other diseases. Tonsillectomy.

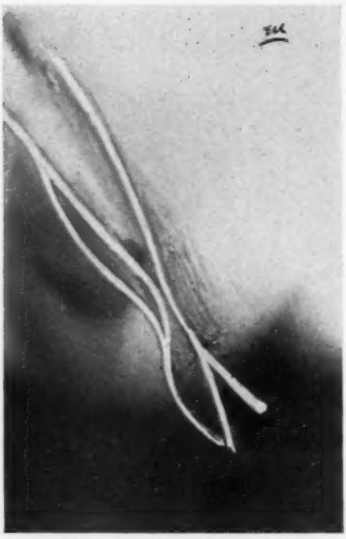


Fig. 6. Sister C. Retouched X-ray film with the diagnostic bag in place. It shows a tubular narrowing of the crural canal and a twist of the terminal portion of the esophagus to the left. In this case the bag did not accomplish any dilatation.

The patient has not been perfectly well for several years. She has had recurrent stomach distress without any particular relation to meals. This was relieved by Seidlitz powders. The patient began to feel run down and to lose weight six months ago, and has considered herself really ill since November.

Present Illness: The patient has had difficulty in swallowing for the past four weeks and has been fed by enema during this time. She vomits after swallowing and states that even liquids fail to go through. She has pain in the left hypochondrium, which does not radiate. There has been no hematemesis. The patient feels that she has lost weight but does not know how much.

There is no cough, no expectoration, no palpitation, and no ankle edema. The bowels are constipated.

Physical Examination: The head is negative. The chest, symmetrical with expansion equal on both sides; resonant throughout. Breath sounds normal. No rales. Heart apparently not enlarged. The sounds are of good quality, and the rate is regular. There are no murmurs. The blood pressure is 110/90.

Dec. 21, the patient was taken to X-ray this morning and under the fluoroscope the diaphragm was seen to move equally. There was no enlargement of the heart. The chest was negative. The barium was held up at the cardia. After 30 seconds it passed into the stomach. The terminal portion of the esophagus was of even outline. There was nothing suggestive of malignancy (see Fig. 6).

Esophagoscopy: At the cardia there was a symmetrical narrowing of the lumen but no growth. The right crus appeared thickened. A No. 24 French nonopaque bougie passed through the narrowing without much resistance and a duodenal tube was carried through the esophagoscope into the stomach. The feeding tube was brought out through the nose. The tube remained in four days and then was vomited up.

Dec. 26, under fluoroscopic vision the barium was seen to halt in the lower third of the esophagus, but with a Seidlitz powder it passed readily. A No. 30 bougie with flexible finder passed without difficulty. Dec. 27, since dilatation yesterday, the patient swallows much better and is taking fluids without any difficulty. Dec. 31, a No. 24 bougie with flexible finder passed yesterday without difficulty. Jan. 1, 1936, the patient is not taking fluids well. Jan 7, the patient was taken to the X-ray room again and under the fluoroscope the diagnostic bag was passed, and the stricture dilated with ½ lb. pressure. The patient took fluids better following the dilatation. The X-ray film of the bag in place showed a long tubular narrowing of the crural canal. Jan. 9, bag passed and dilated with 1 lb. pressure. Jan. 11, the patient ran a slight temperature following the dilatation. Jan. 16, esophagus dilated with bag. The narrowing of the crural canal did not dilate. Jan. 18, bag did not pass through the stricture. Jan. 26, a No. 44 French bougie passed easily. The bag was not tried. Patient has lost three pounds in weight. Jan. 27, patient is discontented. Discharged to nearby convent to report weekly.

Readmitted Feb. 18. *Interval History:* For about one and one-half weeks after discharge from the hospital the patient

was able to swallow liquids and solid food when she again had difficulty and at the present time has trouble retaining liquids unless taken in very small amounts.

The patient has lost 12 pounds. She looks bad generally and is very despondent. She vomits most of her liquid feedings. Feb. 21, a No. 34 nonopaque bougie was passed under the fluoroscope. Then the bag was tried. The metal finder passed but the bag did not. A feeding tube was passed into the stomach with some difficulty and the patient had a feeding every two hours. Feb. 22, the patient vomited the stomach tube yesterday. The tube was reintroduced and the stomach washed out. There was no vomiting. The patient has held everything on her stomach since then; however, she seems weak. Feb. 24, the patient has taken feedings through the tube with no regurgitation. The tube feedings were reduced in number, however, on account of diarrhea. The patient looks bad. Feb. 25, for one week the patient has had vomiting, necessitating tube feeding. For several days she has had abdominal distention and cramps and for the past two days diarrhea starting with an enema.

The chest is negative. The abdomen is very distended and tympanitic. The extremeties are normal.

Remembering the nurse in whose case dilatation did not give the usual result and who had mediastinitis and tubercular pleurisy after dilatation, I asked for a medical consultation, suggesting that tuberculosis be considered. Dr. Drake and Dr. Jones then saw the patient.

Medical Consultations: Feb. 25. "It may be possible that the patient has a stenosing peptic ulcer of the esophagus, or possibly a mediastinal adenitis with stricture of neighboring esophagus, although there is no X-ray evidence of this.

"Abdominal tuberculosis seems to me a distinct possibility in view of the low-grade fever, rapid pulse, anemia, and cachexia. The patient does not have pernicious anemia."

Feb. 27. "The abdomen is generally tender and resistant and has a doughy feel. No peristaltic activity is heard. Dr. MacMillan states he has seen calcified abdominal glands and that Dr. Mosher thinks tuberculosis could explain the stricture.

"I feel that this is a case of tubercular peritonitis with involvement of the lymph glands about the esophagus, secondary to the involvement of the mesenteric lymph nodes. The

prognosis of a plastic tubercular peritonitis is hopeless when generalized and advanced." — Dr. T. C. Drake.

Feb. 27. "The whole picture to me seems one of tuberculosis with a tubercular mesenteric adenitis, possibly a tubercular colitis, and in addition periesophageal involvement. The prognosis is very grave. I see no indication for surgery. I would attempt to control her symptoms with some opium preparation and give her frequent small feedings. If there is no improvement in a week, I would consider sending her home." — Dr. Chester M. Jones.

Feb. 29, discharged to home convent.

On May 16 the following letter was received from Dr. F. T. Hill, of Waterville:

"Dear Dr. Mosher:

"In re: Sister M. C.

"I saw this patient the day after her return from the infirmary, in consultation with Dr. Bisson and Dr. Gousse, at the convent. At this time she was tremendously dehydrated and the abdomen swollen sufficiently to give the impression of a six months' pregnancy. It was very doughy, and seemed quite characteristic of a tubercular abdomen. She had taken about 15 cc. of fluid that day. I advised intravenous glucose and saline, with consideration of doing a gastrostomy when her condition should warrant. During the next few days she became more and more obviously a case of abdominal tuberculosis. An operation was performed to drain and ventilate the abdomen, rather than a gastrostomy. Two and one-half gallons of fluid were obtained from her belly. The intestines and mesentery were reported as studded with small tubercular ulcerations, and several small discrete, hard nodules were felt in the region of the duodenum. Since operation, she has been reported as improving quite markedly, and is now taking a soft solid diet without any difficulty in swallowing. It would seem as though gastrostomy would not be needed, and that possibly a large part of her obstruction was due to pressure of the tubercular process on the lower esophagus. If gastrostomy were done, I had planned to be present and try to get a view of the cardia from below by retrograde endoscopy. I will keep you informed of her subsequent progress. Sincerely yours,

"Dr. Frederick T. Hill."

SUMMARY.

The finding in this case was a long tubular narrowing at the lower end of the esophagus. A No. 30 flexible finder passed readily and later the bag also passed easily. The bag was used regularly for a considerable period, but little dilatation resulted. Finally, the finder would pass into the stomach, but the bag would not follow it. In other words, the fibrosis did not yield to dilatation with the bag as usually happens in the ordinary case of fibrosis of the terminal portion of the esophagus, in fact, the narrowing increased.

For a time the patient swallowed better and then gradually dropped back, and on her second admission it was found that she had lost 12 pounds in weight. For one and one-half weeks after discharge from the hospital she was able to swallow liquids and solid foods and then the difficulty in swallowing returned and she could retain liquids only when taken in small amounts. She was extremely despondent.

A feeding tube was put in place with some difficulty. This was vomited out the next day and reintroduced. Two days later the progress note says: "The patient is taking feedings through the tube. There is no regurgitation. The tube feedings were reduced in number on account of diarrhea. The patient looks bad."

The patient was seen by Dr. Jones and Dr. Drake who made a provisional diagnosis of tubercular peritonitis and gave a poor prognosis. On Feb. 29 the patient was discharged to her home convent.

COMMENTS.

From the first this case did not run true to form. The tubular narrowing at the terminal portion of the esophagus was unusually long and the barium lines on the diagnostic bag suggested a twist to the left. With the use of the bag, little or no dilatation could be seen under the fluoroscope. What dilatation was accomplished was of but temporary benefit; in fact, it seemed to irritate rather than help. After a short period of improvement it was necessary to resort to a feeding tube a second time in order to control the vomiting.

Finally, diarrhea and swelling of the abdomen appeared. At operation the intestines and the mesentery were found to

be studded with tubercular ulcerations. A large amount of fluid was evacuated.

At some of the examinations marked peristalsis was seen in the esophagus. This raised the question of ulcer, although none was seen at the esophagoscopy; however, considering the findings at operation, it seems probable that there have been ulcerations at the terminal portion of the esophagus at some time and that they were the cause of the tubular narrowing. Further, the fact that the evacuation of two and one-half gallons of fluid from the abdomen was followed by marked improvement in swallowing, raises the question whether or not the pressure of the fluid did not play a part in the esophageal obstruction.

H. D., age 33 years. Mattapan Sanatorium, Mattapan, Mass. Patient came to hospital bearing a letter from the Mattapan Sanatorium dated Aug. 28, 1935. The letter reads as follows:

"To Whom It May Concern:

"This is to certify that Mr. H. D. is a patient at the Boston Sanatorium with a diagnosis of pulmonary tuberculosis.

"He also had a neptrectomy and ureterectomy (left side) in May, 1935, at the Peter Bent Brigham Hospital. He has also been under treatment at Dr. Chevalier Jackson's Clinic.

"This patient will be under treatment for some time.

"Dr. F. H. Hunt, Resident Medical Officer."

The patient's chief complaint was regurgitation of food after eating. On lying down he was much troubled with regurgitation.

Aug. 19. Fluoroscopic examination. No plates. Fluid level at the arch of the aorta. The esophagus is moderately dilated. It ends in the typical awl-like point. No barium passed from esophagus into the stomach and Seidlitz powder was given. Flexible finder No. 30 passed easily.

The patient has been under treatment for seven months. Before passing bougies it was necessary for the patient to empty his esophagus by retching or have the esophagus washed out. The patient came for bouginage at first at weekly intervals.

By Sept. 17, the patient gained six pounds. The diagnostic bag was not passed on account of the diagnosis of tuberculosis. By Oct. 1, the patient gained 10 pounds. A mercury bougie No. 53 French was passed. Then the patient was given a bougie and taught to pass it himself. By November, the patient was passing his own bougie daily and eating everything. The fluid level was two inches below the arch of the aorta. The barium did not enter the stomach until forced by a Seidlitz powder. By January the patient eats everything but orange juice. There is no vomiting. The patient has no trouble in swallowing and passes the bougie daily. March 19, the patient is still passing the bougie and still has a fluid level at the arch of the aorta.

Impression: On account of the near tragedy which happened to Miss K. S., the graduate nurse, who developed tubercular pleurisy after dilatation with the bag, the bag was not passed at any time. From the history of pulmonary tuberculosis and the old history of tuberculosis of the kidney, there was ample cause for a fibrotic narrowing of the terminal portion of the esophagus. He was regurgitating all food, which was especially annoying at night when he attempted to lie down. The only relief he obtained was to induce vomiting by pushing his finger down his throat. At all times, even after seven months of treatment, the fluid level remained practically at the arch of the aorta and no fluid entered the stomach from the esophagus during the fluoroscopic examination unless forced down by the patient's taking a Seidlitz powder. In other words, the dilatation brought about by the bougies was more or less temporary. For a while it was necessary to have the patient empty his esophagus by induced gagging or by washing it out before a bougie could be passed comfortably.

Unlike the other two cases, this patient had the usual optimism of the tubercular patient. In the nurse and the Sister of Charity the reverse was true; both were extremely depressed. In the case of the Sister, particularly, she was so low spirited that I wrongly accused her of not being co-operative. As it turned out, she was more sick than it appeared to us and there was ample reason in her toxemia for her depression.

243 Charles Street.

A SUGGESTED ROUTINE TECHNIQUE FOR EMERGENCY TRACHEOTOMY.*

DR. WM. B. CHAMBERLIN, Cleveland.

Early one morning several years ago I received a telephone call from a medical colleague in a neighboring village stating that his child, a boy aged 6 years, was suffering from a dyspnea, which had been increasing in severity for the past few hours, and asking if I could see the child at my office. He thought that the dyspnea was of laryngeal origin and suggested the possibility of a foreign body, though there was no definite history of the child having had anything in his mouth. Neither was there any history of any acute inflammatory process, as the child had gone to bed apparently in perfect health and there was no fever. I advised instead that the father take the child immediately to the hospital and that I would meet them in the operating room. In the meantime I telephoned my assistant to make the usual preparations for an immediate bronchoscopy and for an emergency tracheotomy as well.

On entering the operating room some 45 minutes later I saw the child sitting on the operating table, both hands grasping the sides of the table in the position of extreme orthopnea, and gasping for breath. There was no cyanosis and cursory examination of the pharynx showed no acute inflammation. I realized that a tragedy might be imminent and stationed my assistant, a thoroughly competent surgeon, beside the table, prepared to perform an emergency tracheotomy. I then took my position at the head of the table, laryngoscope in hand, prepared to remove as quickly as possible the foreign body from the larynx, if such proved to be the cause of the laryngeal obstruction. The patient was then lowered to the recumbent position. No sooner was this position assumed than the breathing immediately ceased and before the larynx could be examined or the trachea opened the child was dead.

*Read at the fifty-eighth annual meeting of the American Laryngological Association, Detroit, May 25, 1936.

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A limited autopsy was at once performed. No foreign body was found, the sole cause of the laryngeal obstruction being apparently an acute edema of the glottis, etiology unknown.

This accident and tragedy made a deep impression upon me. Many times during the day and frequently at night have I gone over the details of this scene and tried to work out some plan whereby such a tragedy might be circumvented in the future. And gradually through the years there has been evolved the technique for emergency tracheotomy which we have come to use routinely in our service at the Lakeside Hospital.

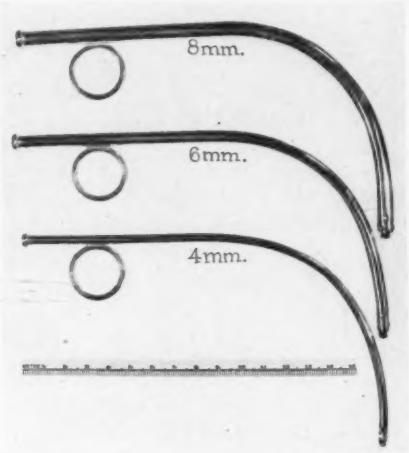

Fig. 1. Three sizes of Mosher tubes, external diameter 4, 6 and 8 mm.

Tracheotomy is performed for two reasons only: *1.* as a preliminary to certain operations on the larynx in order to *a.* allow free respiration during the operation and, above all, *b.* to prevent the passage of blood during the operation into the trachea and lungs; *2.* to provide a bypass for the passage of air as the result of acute or chronic laryngeal obstruction.

In the former case it is a deliberate operation performed universally, as I take it, under local anesthesia. If the laryngeal obstruction is of long standing the operation is also deliberate; no undue haste is essential and no untoward results

as a rule follow. But a tracheotomy for the relief of sudden and acute laryngeal obstruction is a far different matter. The surgeon, realizing that every minute is precious and that he may be confronted at any moment with a tragedy, is forced to operate under the most trying circumstances. The result is that the vast majority of such operations are poorly performed and that if a fatality does not immediately follow it not infrequently follows later as the result of a faulty technique. In many instances such haste has been averted by

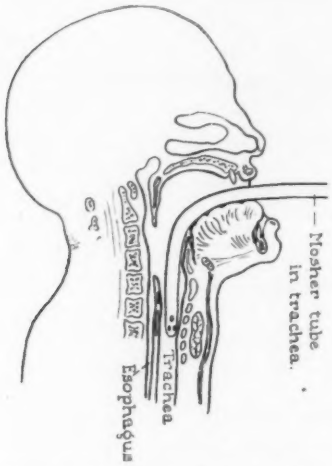

Fig. 2. Tube is shown in position in larynx and upper part of trachea.

the use of an intubation tube or the introduction of a bronchoscope. The objection to the former is the difficulty of introduction and also that the lumen of the tube is far too small. The first objection, the difficulty of introduction, applies also to the bronchoscope. To my mind the answer to our problem, as well as the answer to both of the foregoing objections, has been given by the so-called life saver of Mosher, with which you are all familiar. Its ease of introduction in either the erect or reclining posture leaves nothing to be desired, while its lumen gives ample space for free respiration while the tracheotomy is being performed. The

three graduated sizes of tube also give ample airway for any size of larynx.

The Mosher tube may be introduced with the patient in the upright or recumbent position. The technique in the main is the same as that used in the introduction of an intubation tube, except that there is no introducer to be removed. The forefinger of the left hand is carried over the base of the tongue until the tip of the epiglottis is felt. In children it is much better to feel for the arytenoids, which are distinguished

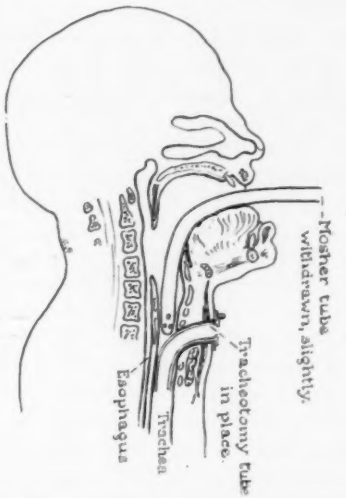

Fig. 3. Tube is slightly withdrawn, showing tracheotomy tube in position.

as two small lumps on the flexor surface of the index finger. Incidentally, this prevents the tube from slipping into the esophagus. The tube is now carried along the flexor surface of the index finger until its point is directly above the opening of the larynx. The handle or proximal end is now elevated and the tip, guided by the finger, pushed gently through the larynx and into the trachea.

After the tube has been introduced it is held in position by a nurse or assistant to see that it is neither coughed out

or displaced. The patient is then placed in the usual position for the tracheotomy, with a small pillow under the shoulders and neck and the head thrown well back, the tube being still held in position and the head in direct line with the body and in the true vertical position. Meanwhile, the respiration is perfectly free. Older patients are given an appropriate, though small, dose of morphine. With children morphine is not used. The skin and tissues about the trachea are now infiltrated with 0.5 per cent solution of procaine, to the drachm of which one or two drops of epinephrin are added. As a rule this will take care of all bleeding, though occasionally some larger vessels will have to be ligated. The trachea over the upper four or five rings is now rapidly skeletonized largely by blunt dissection, the incision being either transverse or vertical, as suits the whim of the individual operator. Before the trachea is opened all bleeding is carefully controlled and the field inspected to see that it is perfectly dry. A few drops of a 2 to 4 per cent solution of cocaine are now introduced into the lumen of the trachea by means of a fine hypodermic needle passed between the tracheal rings. This procedure was first recommended, I think, by Dr. D. Crosby Greene, of this Association, and later so strongly advocated by Sir St. Clair Thomson in his address entitled, "Tranquil Tracheotomy." Only those who have witnessed the opening of the trachea without its use can appreciate its value. A slight paroxysm of coughing usually follows the introduction of the cocaine. This coughing may also cause some bleeding not previously apparent. After an interval of four or five minutes to allow the cocaine to take effect, the trachea is opened either between the rings or by cutting directly across one of them. In older patients where larger tubes are used it is usually advisable to resect a portion of the ring to prevent undue pressure from the tube. This piece should be firmly grasped with forceps or hemostat to see that it is not aspirated. A blunt hook is then inserted through the opening and the trachea drawn slightly upward. The old distinction of high, median or low tracheotomy at the present time has little or no meaning or value. The isthmus of the thyroid may be pushed upward or downward, as the case may be, but if neither of these procedures is convenient it is divided and the cut ends carefully ligated. When the trachea is opened the convex surface of the Mosher tube, rather than the end, is usually brought into view.

The tube is now slightly withdrawn until the end appears above the first hook. Two other hooks are now inserted, one on either side, the tracheotomy tube placed in position and firmly tied with tapes behind the neck. Two or more skin sutures may be necessary, also a small drain immediately above and below the tube to take care of superficial and late bleeding. The Mosher tube is then withdrawn.

A recent case may serve as a favorable illustration as to the value of the operation just described. During the past winter I was called at 2:30 A. M. to see a patient suffering with acute laryngeal obstruction due to edema of the glottis. Neither the edema or the dyspnea were extreme, but it was thought best to remove him to the hospital. On visiting the patient again at 8 A. M., I found him gasping for breath and a crisis apparently imminent. The large size Mosher tube was quickly introduced and held in position while he was removed to the operating room. His dyspnea was immediately relieved and the tracheotomy performed with no haste or anxiety and, above all, with no discomfort to the patient. Although he was still convalescing from a recent resection of the prostate his recovery was uneventful.

A similar experience occurred in the practice of my colleague, Dr. C. E. Pitkin. He was called at 5:30 in the morning to see a child of 4 years who was suffering from acute laryngeal obstruction. As the dyspnea was not extreme he followed the child to the hospital, taking the precaution to put a Mosher tube in his pocket. On reaching the accident ward shortly after the arrival of the patient he found him in *extremis* and just about breathing his last. The Mosher tube was immediately inserted, the dyspnea relieved and the tracheotomy performed under local anesthesia.

Possibly I am still among the few who consider a tracheotomy on a youngster with a short, fat, thick neck a difficult operation. But I shall continue to think so, thereby acknowl edging no doubt my surgical limitations. The old stab operation is still described in many textbooks and the tracheotomy tube with trocar obturator frequently illustrated. Personally I have never performed this operation, nor have I seen it performed. I trust that I never shall, though I can conceive of situations where it might become obligatory.

Sir St. Clair Thomson has enumerated the following accidents which may attend a tracheotomy:

1. The opening in the trachea may not be made in the midline.

2. If the opening is incomplete, the cannula may make a false passage for itself beneath the mucous membrane.

3. If the knife is not held carefully, it may not only open into the windpipe but traverse the opposite side.

4. The trachea may be entirely incised and the esophagus or some other structure opened by mistake.

5. The cannula may fail to enter the wound in the trachea and may slip down in front of it and below the fascia.

6. The tube may be of the wrong shape or size, or its orifice may become occluded by membrane or blood.

7. There may be difficulty in introducing the cannula. Clumsy efforts should never be made but the trachea held open with two sharp hooks; I often use three.

CONCLUSIONS.

1. An emergency tracheotomy is always a trying operation.

2. As a result it is frequently hastily and often poorly performed.

3. The foregoing technique is suggested as a routine procedure in order that the difficulties attending and following the older procedures may be avoided.

Carnegie Medical Building.

AMERICAN OTOLOGICAL SOCIETY.

Sixty-Ninth Annual Meeting, May 28 and 29, 1936.

(Continued from September issue.)

The statements of facts and the recommendations made in the report, which was mailed to all members of the Society in advance of the meeting, are presented under six major headings, subdivided as follows:

I. INTRODUCTION.

II. SUITABLE SOURCES OF SOUND FOR BONE CONDUCTION TESTS.

- a. For threshold tests.
- b. For tests above threshold.

III. THE EFFECT OF ROOM NOISE UPON THE ACCURACY OF TESTS OF HEARING.

IV. THE USE OF MASKING IN CLINICAL TESTS OF HEARING.

- a. Why use masking?
- b. Which ear should be masked?
- c. How can one ear be masked?
- d. Methods of producing satisfactory masking sounds.

V. SPECIAL POINTS ABOUT THE USE OF TUNING FORKS.

- a. Technique of handling fork after it is struck.
- b. Activation of tuning forks. Standard blows.
- c. Damaged and otherwise unsuitable forks.
- d. Where place the stem of the fork?
- e. Calibration of tuning forks.

VI. SPECIAL POINTS ABOUT THE USE OF AUDIOMETERS WITH BONE CONDUCTION RECEIVERS.

Of the positive recommendations of the Committee the most important are:

1. That all tests of hearing acuity be made in quiet surroundings.

2. That tests of the hearing by bone conduction be made for as great a range of frequencies as is possible with the available instruments, always with due regard to the possible sources of errors.

3. That suitable masking sounds be employed routinely while making all tests of the acuity of hearing by bone conduction. Practicable methods of producing satisfactory masking sounds are described.

1. Anatomical, Physiological and Pathological Observations. Stacy R. Guild, Ph.D.

After a brief review, by means of charts (lantern slides), of the anatomical structures primarily concerned with hearing, and of the possible pathways by which sound waves may reach the organ of Corti during hearing by bone conduction, the speaker presented evidence that one of the optional routes is of more importance than the others. His studies of human temporal bone sections, from patients whose hearing had been examined, reveal a correlation between bone conduction time (the data with respect to bone conduction were obtained with a steel tuning fork of the frequency of 512 double vibrations per second) and the presence, or absence, of microfractures of the short osseous trabeculae that connect the medial end of the posterior wall of the external auditory canal to the inferolateral surface of the prominence of the horizontal semicircular canal. Microfractures of these trabeculae, with fibrous union of the fractures, are present in the sections from patients who had a moderate or a marked shortening of bone conduction time and normal, or but slightly impaired, hearing by air conduction of the sound; such fractures are not present, or are incomplete, in the sections from patients with a normal relationship between the hearing by air and by bone conduction, or from patients with good hearing by bone conduction and an impaired hearing by air conduction.

Hearing by bone conduction undoubtedly is impaired by lesions of the organ of Corti, the cochlear nerve or the auditory pathways of the central nervous system. But, so far as revealed by the speaker's studies, such lesions always impair the hearing by air conduction as much as that by bone conduction. The only lesions of the inner or of the middle ear that have been observed that are peculiar to ears with a disproportionate impairment of hearing by bone conduction are the microfractures of the osseous trabeculae above described.

An hypothetical explanation of the association of these facts is advanced. It is: That sound waves entering the intralabyrinthine fluids from that part of the otic capsule to which these trabeculae are attached are more effective in stimulating the organ of Corti than are sound waves which are transmitted to the intralabyrinthine fluids through other parts of the otic capsule. Sound waves via the described trabeculae are transmitted to the perilymph from the posterolateral wall of the vestibule, at a region adjacent to the footplate of the stapes, and almost opposite to the opening of the scala vestibuli into the vestibule. The facts warrant the suggestion that this is the direction from which sound waves most effectively cause stimulation of the organ of Corti.

The suggestion is also warranted that poor hearing by bone conduction does not necessarily signify a "nerve deafness"; i. e., the lesion responsible for the impairment may be in the structures that transmit bone-conducted sound waves to the inner ear.

Animal experiments, designed to test the possibility that the bone conduction route for sound waves may be primarily via the so-called osseotympanic route, were also reported. The Wever-Bray phenomenon was utilized to measure the relative intensities of the sound waves reaching the organ of Corti before and after interruption of the ossicular chain by an operative removal of the lenticular process of the incus. Sound waves from the stems of tuning forks and from a hearing aid type of bone conduction receiver, attached to an audiometer, were transmitted to the heads of anesthetized cats through a wooden rod 12 inches long, the end of which was held against the skin over the lateral end of the occipital ridge. The changes in electrical potential produced in the inner ear were picked up by an electrode in the region of the round window niche, suitably amplified and converted into sound waves by a telephone receiver. For the frequencies of 256 and 512 double vibrations per second the bone conduction thresholds were not impaired by removal of the lenticular process of the incus; but they were slightly impaired, on the average, for the frequencies of 1024 and 2048 double vibrations per second.

As would be expected, this lesion regularly caused great impairment of the thresholds for air-conducted sounds of all frequencies. These experiments indicate that at least for the lower frequencies the ones commonly used in clinical examinations of hearing by bone conduction, the osseous pathway to the inner ear is of much more importance than is the osseotympanic route.

The Best Ear Average for AC and BC of Individuals Who Consider Their Hearing Normal. Dr. E. P. Fowler and Dr. E. P. Fowler, Jr.

In studying the audiograms of 1064 hospital patients it was found that those who denied any hearing defect were often somewhat hard of hearing. Averages taken of the best ears of these individuals showed good bone conduction for the lower tone ranges for all decades. In the later decades there was in addition a high tone loss for BC as well as for AC. Many of the older individuals showed remarkably good hearing. Types of illness were studied and the rheumatic fever group has the worst average normal hearing. The importance of testing the air and bone conduction, using the same units for each, was emphasized, as well as the fact that in all individuals upper respiratory infections are important because they cause or increase lesions which cause deafness in the most used lower tone ranges, not only in children but also in the later decades.

Pathologic Changes in the Middle Ear of Patients with Normal Hearing and of Patients with a Conductive Type of Deafness. Dr. LeRoy M. Polvogt and Dr. John E. Bordley.

The object of this study is to try to determine the common types and locations of the lesions in the temporal bone that are associated with a conductive type of deafness and a negative Rinne.

First, before attempting to analyze these lesions in ears from patients whose hearing is better by bone than by air conduction, it is necessary to become familiar with the pathologic changes found in the middle ear of patients with normal hearing.

We studied the records of some 950 tested ears; from this number only 63 ears met the requirements for the normal group, and 20 ears met the requirements for the group in which bone conduction time was better than air.

The results of a comparative study of the histologic lesions in the middle and inner ears of patients with normal hearing and those with a conductive type of deafness and a negative Rinne are as follows:

1. The most striking difference is the absence of any lesion of the ossicles in the group with normal hearing, and the presence of one or more lesions involving the ossicular chain in every ear in the group with impaired hearing.

2. We believe when the Rinne test is negative it is the result of one or more lesions interfering with the normal movements of the ossicular chain. This causes a reduction of the air conduction time without changing the time of bone conduction.

3. The pathologic specimens on which these studies are based did not include the entire Eustachian tube, so we have no information about tubal lesions in patients with normal or impaired hearing.

Skull Resonance. Dr. Douglas Macfarlan.

It is obvious that by investigating bone conduction hearing we seek to determine the true functioning of the end-organ of hearing unhampered by effects that might take place where the transmission apparatus of the middle ear has been impaired.

In testing bone conduction not only is sound conducted to the cochlea by direct bone transmission but also a general skull resonance is set up which

may considerably alter test findings. These effects are due: 1. to lack of uniform density, size and shape of the two halves of the skull; and 2. filling of the sinuses, particularly the frontals and antra.

Lateralization of the sound from midline points (the Weber test) is greatly influenced by factors affecting skull resonance, and misleading conclusions may occur if resonance is not considered.

The contralateral reference of sound has not been satisfactorily explained. It does not occur with any great regularity or dependability. (Clinically, a fork placed on one parietal eminence should be heard in the opposite ear.)

The classical finding of sound reference to the good ear in nerve deafness, and to the bad ear in catarrhal deafness, is not invariably reliable. In acute mastoiditis there is frequently found the Weber-Schwabach paradox of MacKenzie, in which BC lengthened on the affected side yet reference from the midline is toward the good ear.

Lateralization from the midline may vary with the pitch used (alternating Weber, Macfarlan).

In bilateral otitis the value of lateralization is dictinctly doubtful. Techniques for testing skull resonance are described.

To minimize the absorption of fork sound by resonance when testing by bone conduction, the on-and-off method of applying the fork (Fowler) is urgently recommended. This technique allows the patient to have contrasts of sound intensity, and reduces the tendency to develop tone memory while testing.

Bone Conduction, Vibration and Electrical Stimulation. Dr. Hallowell Davis.

The relation between bone conduction and the vibration sense is considered. The high differential sensitivity for pitch, similar to that of normal hearing and far superior to that of the vibration sense, shows that bone conduction stimulates the true auditory mechanism.

Experiments involving direct electrical stimulation of the auditory mechanism are reviewed, and here it appears that, although direct stimulation of the auditory nerve is possible, nevertheless a normal inner ear is essential for pitch discrimination. This renders impossible the use of such electrical stimulation as an "artificial ear" in cases involving loss of the organ of Corti. Bone conduction and electrical stimulation are useful as diagnostic tools or as aids to hearing only as alternative avenues of approach to the inner ear, which is essential for useful hearing.

Clinical Observations. Dr. George E. Shambaugh, Jr.

Since the advent of the audiometer there has been renewed interest in hearing tests and in evaluating the audiometer compared to tuning fork tests, particularly with regard to bone conduction. The standard audiogram cannot be used to diagnose the type of deafness since the shape of the curve is not characteristic. The standard curve supplemented by the bone conduction curve is not sufficient because the standard curve has an element of bone conduction from the usual receivers, giving a false picture of the hearing for low tones in conduction deafness. Pure air conduction must be used, but this is inaccurate and difficult to make. For practical purposes the tuning fork tests are quicker and easier to make, and more accurate for diagnosing the type of deafness.

The prolongation of bone conduction found in conduction deafness is due principally to the exclusion of outside sounds by the lesion, so that in a soundproof room the Schwabach is normal in lesions of the conducting apparatus. The Schwabach test, therefore, should not be done in a soundproof

room. Moreover, the noisier the room the more clearly will a prolonged Schwabach be evident in cases of early conduction deafness.

It has generally been assumed that different kinds of conduction deafness give the same hearing tests provided the degree of deafness is equal. This is not true. Where the middle ear cavity is filled with fluid, as in acute suppurative or catarrhal otitis media, there is an impairment for the entire tone range, the high tones nearly as much as the low. In lesions involving only the oval window, such as otosclerosis or traumatic perforation of the drum membrane, the loss of hearing involves predominantly the low tones which are markedly depressed with little or no lowering of the high tones. This difference in the hearing tests in the two types of bone conduction deafness can best be explained by considering the mechanism of hearing by bone conduction.

While most cases of conduction deafness are easily diagnosed by the tuning fork tests, one type of nerve deafness that involves chiefly the low tones may easily be confused with conduction deafness. This is the type associated with diplacusis, often with vertigo, where the lesion is probably an inflammatory exudate in the labyrinth, an exudative labyrinthitis. The strongly positive Rinne and shortened Schwabach serve to differentiate this type of low tone nerve deafness from conduction deafness.

Clinical Observations on Bone Conduction. Dr. W. J. McNally, Dr. T. C. Erickson, Dr. R. Scott-Moncrieff and Dr. D. L. Reeves.

The object of this research was to investigate bone conduction acuity in a series of patients suffering from inner ear or proven cochlear nerve lesions, such as acoustic nerve or other intracranial tumors.

Much care was exercised in selecting the instruments for testing bone conduction. We were guided in our selection by the report of the Committee on Hearing Tests, of the Royal Society of Medicine, London, 1933.

A suitable routine of examination was adopted, and was used in all cases except where the preliminary hearing tests had been done before this investigation was undertaken. Insofar as possible, a complete hearing test was done before and after any operative procedure. The complete examination consisted of:

a. A careful history and inspection of the ears, nose and throat.

b. An audiogram (2A Western Electric Audiometer).

c. Tests with the audiometer bone conduction apparatus of bone conduction, and absolute bone conduction for the notes 256 d.v. and 512 d.v.

d. Tests with a tuning fork (512 d.v.) of air conduction.

e. Tests with a von Struychen monochord of air conduction and bone conduction.

f. A vestibular examination.

A standard length of vibration of the tuning fork was obtained by using a metal clamp which was removed suddenly, it having slightly compressed the prongs. The vibration time was recorded in seconds.

When there was a difference between the hearing in the two ears, the non-tested ear was masked with either a continuous current of air, an electric buzzer or with a Barany noise apparatus.

The patients examined fall into five groups for descriptive purposes:

Group 1: Thirteen cases were examined before and after encephalography and ventriculography to determine the effect of possible changes of intracranial pressure upon the hearing. A change in hearing was not noted in any case.

Group 2: Fourteen patients had varying quantities of cerebral tissue removed from different areas, usually because of epilepsy or trauma. As noted by previous observers, it was found that large portions of cerebral tissue may be removed from one side without causing any permanent hearing damage.

Group 3: There were six cases of cerebellar tumor. When the lesion was confined to the cerebellum there was very little change in hearing.

Group 4: In nine cases of cerebral tumor there was no instance of hearing loss attributable to the brain lesion.

Group 5: There were four cases of perineural fibroblastoma of the VIIIth nerve and one of Vth nerve. Usually there was almost complete deafness on the involved side but it required masking to demonstrate the true condition. In one case where the VIIIth nerve was not completely damaged, the hearing actually improved postoperatively on the diseased side.

Group 6: There are five miscellaneous cases in this group, including an VIIIth nerve section for Meniere's syndrome, and one case of third ventricle tumor which showed a loss of high tones after removal of the tumor.

CONCLUSIONS.

Of the three methods used to test bone conduction (the audiometer BC attachment, the tuning fork and the monochord), one did not prove any more reliable than the others.

Whether bone conduction was tested on the audiometer for the note 512 d.v. or 256 d.v. made very little difference.

In four patients, each of whom apparently had one totally deaf ear, the bone was greater in the poorer hearing ear.

The Weber test proved to be very misleading.

The monochord served as a valuable check upon the audiometer but it is very difficult to mask out its effect from the nontested ear. The continuous current of air proved to be the most effective masking apparatus for the monochord.

Our experiments would suggest that the most accurate method of testing hearing at present is to combine the use of the audiometer, the tuning forks and the monochord for testing both air and bone conduction, and that masking the nontested ear may be an essential part of the test.

During this investigation 52 patients were examined. In the complete report a short summary will be given of each case.

Discussion of Report on Bone Conduction. Dr. Isidore Friesner.

The report of the Committee on the tests of hearing by bone conduction contains a great deal of important information of such a type that it is difficult for an ordinary practicing otologist and not a physicist to intelligently discuss it.

However, there are some suggestions in the report which I would like to review from the point of view of the practicing otologist. It seems to me that tests of hearing and that, of course, includes tests by bone conduction, have a three-fold purpose. First, and by far the most important, is diagnosis; secondly, adequate records; and finally, for the future perhaps, as a basis for

the selection of that type of hearing aid most suitable to the patient under consideration.

It is particularly with regard to the first purpose of ear tests that I believe we must still keep our feet on the ground and therefore I wondered at the statement "that we do not know how to interpret the information from certain bone conduction tests, etc." We may not be able to explain these phenomena but is not interpreting precisely what we have been doing for years with considerable success? Obviously, tests must be made under proper conditions and in a proper manner but under such circumstances "shortened bone conduction" should have a very definite significance, a significance that has been of great value to all of us in diagnosis, a significance that is quite as important as "slight dullness at the apices" is to the internist.

I am glad, therefore, that the Committee did not attempt the standardization of bone conduction tests. First, because I would be very loath to abandon the tests which have been so useful to me through the years; and secondly, I would be equally unwilling to discard the many records that I have accumulated, from which I have taught, and which have been made on the basis of these tests.

DISCUSSION.

R. L. WEGEL: There is a considerable amount of circumstantial evidence indicating that for any given pitch, the same portion of the auditory nervous tissue is stimulated both by air and bone conduction and probably in the same way. It does not follow that since the tines and shank of a tuning fork are vibrating at the same frequency, the pitch must be the same; yet this is substantially the case. Jones and Knudsen (1930) have shown that acuity by bone conduction bears a strong resemblance to air conduction in the way with which it varies with frequency. This is another of a number of indications that the vibration of all parts is substantially the same in both cases.

If the ear is tightly stopped by depressing the tragus with the finger, the sound from the air is reduced about 35 db. A sound wave striking a material like the head is largely deflected but that which is transmitted into it is in the neigborhood of 35 db. below the level of that in the air. Pending a precise investigation of this problem we may reasonably explore the consequences of the conclusion that on an energy basis the ear is about as efficient in picking up sounds from the bone as from air. In view of the great length of sound waves of acoustic frequency compared with the dimensions of the bony capsule it does not appear useful to think of paths as definable channels through which sound energy flows to the end-organ. It seems more reasonable to assume that stimulation takes place by a bodily vibration or distortion of the capsule or by a modified air conduction. When resolved, this problem will probably be found much simpler than we have been anticipating.

In regard to the testing of bone conduction, there is nothing that can be done with tuning forks which cannot also be done with properly made and properly used electrical vibratory apparatus. This new type of equipment will in fact be capable of much more flexibility and on it must depend further substantial advances in the understanding of bone conduction. In the meantime, there is no reason for the otologist, in his practice, to abandon the tuning forks which he has been using so successfully in diagnosis for years.

(To be continued in a succeeding issue.)

BOOK REVIEW.

Diseases of the Air and Food Passages of Foreign Body Origin. Chevalier Jackson and Chevalier L. Jackson. Philadelphia: W. B. Saunders Co., Aug., 1936. Octavo, pp. 994. Cloth, $12.50.

The reviewer of this new volume by the Jacksons has been able to trace from the very first work,* published in 1907, to the present book, the earnest desire of the authors to pass on all the knowledge and ability that only great capacity for infinite detail can command.

The present book is especially valuable because of the beautiful reproductions of actual foreign bodies as seen by the bronchoscopist through the bronchoscope. This, coupled with a statistical compilation of 3,000 cases, will quickly place in the reader's hands a ready opportunity of surveying a great number of cases and make it possible to assimilate the authors' experiences without great difficulty.

For the many oto-rhino-laryngologists capable of doing bronchoscopies this new volume will be indispensable; it answers many questions and confirms many views already held. As most of the foreign body cases are first seen either by the general practitioner or pediatrist and are frequently not diagnosed, this book, if put in their hands, would do much to prevent foreign body disasters.

The authors recommend bronchoscopic procedures in any case where the possibility or the probability of an intruder cannot be excluded; they clearly show that many times X-ray evidence is practically negative. For those of the profession who occasionally encounter cases which may have a foreign body origin, it will be well to study this book carefully. E. L. M.

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*Tracheo-Bronchoscopy, Esophagoscopy and Gastroscopy. The Laryngoscope Co., St. Louis, Mo., 1907.